VOLUME 49 NUMBER 6

THE LIBRARY OF CONGRESS SERIAL RECORD
JUL 4 1944
Copy 1

ARCHIVES
OF
DERMATOLOGY AND SYPHILOLOGY

EDITORIAL BOARD

HOWARD FOX, Chief Editor
140 East Fifty-Fourth Street, New York 22

C. C. DENNIE, Kansas City, Mo.
HAROLD N. COLE, Cleveland
HERBERT RATTNER, Chicago
FRED D. WEIDMAN, Philadelphia
GEORGE M. MacKEE, New York
C. GUY LANE, Boston

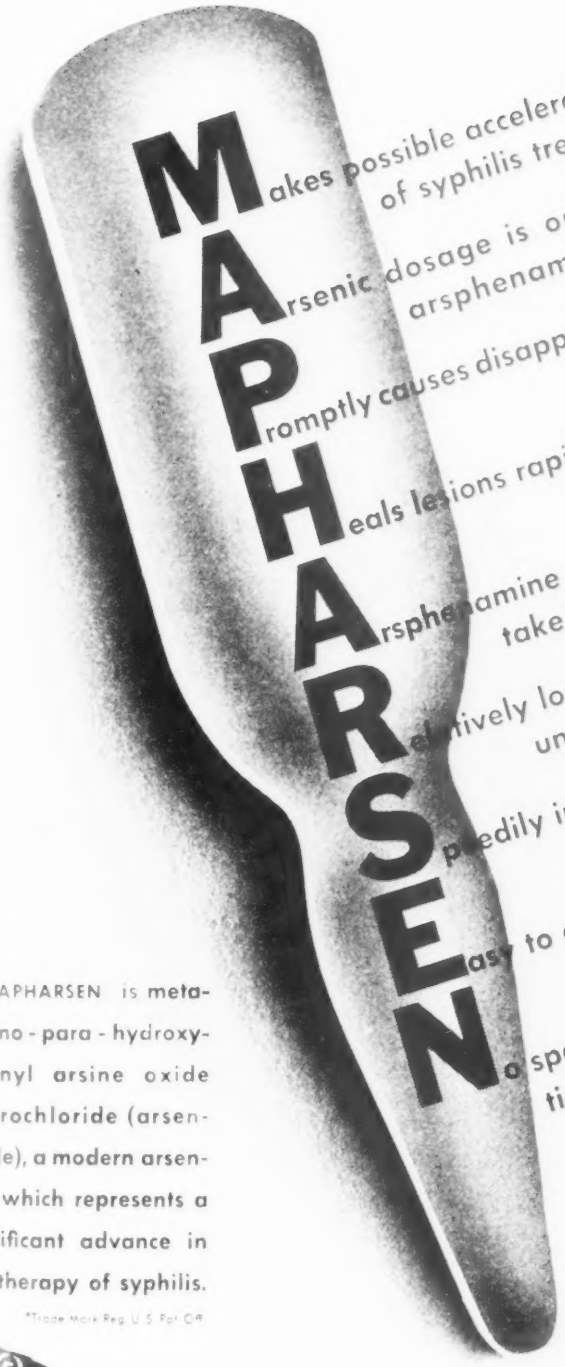
JUNE 1944

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO 10, ILLINOIS. ANNUAL SUBSCRIPTION, $8.00

Entered as Second Class Matter Oct. 30, 1917, at the Postoffice at Chicago, Under the Act of Congress of March 3, 1879

COPYRIGHT, 1944, BY THE AMERICAN MEDICAL ASSOCIATION

JUL -3 1944


MAPHARSEN
Makes possible accelerated forms of syphilis treatment
Arsenic dosage is one-tenth that of arsphenamines
Promptly causes disappearance of spirochetes
Heals lesions rapidly
Arsphenamine intolerant patients can take full doses
Relatively low toxicity . . . fewer untoward reactions
Speedily injected
Easy to administer
No special preparation needed

MAPHARSEN is meta-amino-para-hydroxy-phenyl arsine oxide hydrochloride (arsenoxide), a modern arsenical which represents a significant advance in the therapy of syphilis.

*Trade Mark Reg. U. S. Pat. Off.

Parke, Davis & Company
Detroit 32 Michigan

Luzier Cosmetics and Allergy

Women use cosmetics because they have developed a need for them: they are essential to modern standards of good-grooming and therefore contribute to a sense of well-being. Your patient's appearance, viewed cosmetically, is a factor that deserves your consideration both during hospitalization and convalescence. Cosmetics cannot lift faces, but they certainly perform wonders when it comes to lifting a woman's spirits. Women have an instinctive desire to look pretty and to smell sweet.

Since cosmetics are so universally used it is not to be wondered that they sometimes figure in the field of allergy. That is why when there is a history of allergy we suggest that patch tests be made with those of our products the subject is using or contemplates using. If they test positive, further testing with their constituents is indicated to determine the offending agents. These found, we frequently can modify our formulas to suit the subject's requirements. The patch test is generally considered best for testing cosmetics because it most closely approximates the conditions under which they are normally used.

While our products are free from so-called common cosmetic allergens, such as orris root and rice starch, we feel it should be made clear that any of their normally innocuous ingredients might be allergenic to the allergic individual. It is our practice to write our patrons a letter to this effect when a history of allergy is involved.

It is our experience that many persons with allergic constitutions cannot tolerate scented cosmetics; therefore we routinely recommend and select unscented products when there is a history or suspicion of allergy. This practice is not to imply or suggest that the subject is sensitized to perfume; it is solely to safeguard against the possibility.

In specific cases of allergy or suspected allergy, when the subject is using or contemplates using our products, we are pleased on his request to send her doctor the involved raw materials for patch testing, also such information concerning our products as may have a bearing on the case.

Since in the light of present knowledge it is not possible, save in specific cases, to make non-allergenic cosmetics, we believe the cosmetic requirements of the allergic individual should be considered by her doctor in the light of the formulas and general characteristics of the products she is using or contemplates using.

Luzier's, Inc., Makers of Fine Cosmetics & Perfumes

KANSAS CITY, MISSOURI

More dermatologists buy

Mennen Lather Shave

for their personal use

than any other brand . . .

one third more than

the next leading brand.

Results of a survey among the

Dermatologists on the official

A. M. A. roster

MENNEN

For Surgical Antisepsis

Zephiran Chloride is a germicide of high bactericidal and bacteriostatic potency. In proper dilutions it is nonirritating and relatively nontoxic to tissue cells.

Zephiran Chloride possesses detergent, keratolytic and emulsifying properties, which favor penetration of tissue surfaces, hence removing dirt, skin fats and desquamating skin.

INDICATIONS

Zephiran Chloride is widely employed for skin and mucous membrane antisepsis—for preoperative disinfection of skin, denuded skin and mucous membranes, for vaginal instillation and irrigation, for vesical and urethral irrigation, for wet dressings, for irrigation in eye, ear, nose and throat infections, etc.

HOW SUPPLIED

Zephiran Chloride is available in

TINCTURE 1:1000 Tinted

TINCTURE 1:1000 Stainless

AQUEOUS SOLUTION 1:1000

in 8 ounce and 1 gallon bottles.

Write for informative booklet

WINTHROP CHEMICAL COMPANY, INC.

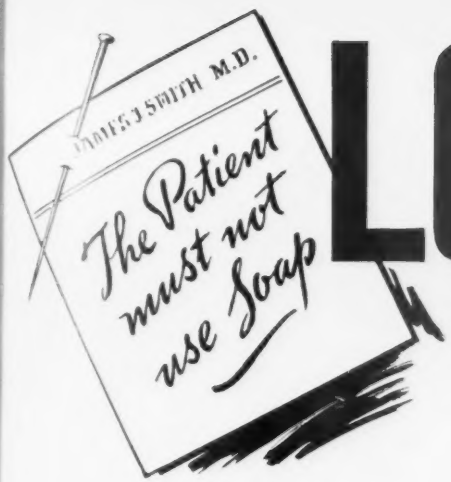
Pharmaceuticals of merit for the physician

NEW YORK 13, N. Y. • WINDSOR, ONT.

ZEPHIRAN CHLORIDE

"Zephiran" Trademark Reg. U.S. Pat. Off. & Canada

Brand of BENZALKONIUM CHLORIDE REFINED



LOWILA

A SOAPLESS DETERGENT IN CONVENIENT CAKE FORM

LOWILA CAKE contains lauryl sulfoacetate diluted in bentonite. The pH approximates that of normal skin (pH 5.0 to 5.5)—it is free of all perfume.

•

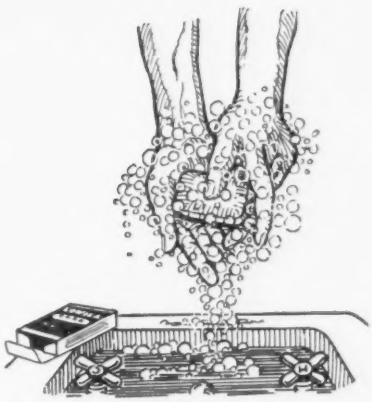
LOWILA CAKE is non-irritating, only mildly defatting and of low sensitizing index; cleanses the skin as satisfactorily as soap—at a cost comparable to that of soap.

•

LOWILA CAKE is an excellent cleanser for general toilet use—including the bath and shaving.

•

SUGGESTED as a SOAP SUBSTITUTE in all conditions due to or aggravated by the use of soap and allied cleansers.



Lowila Supplies
Soft, Creamy Suds

Please Fill In, Clip, and Mail Coupon for Liberal Trial Supply

WESTWOOD PHARMACAL CORP.
1020 Main St., Buffalo 2, N. Y.

Send Lowila Cake for Clinical Trial:

(PLEASE PRINT NAME AND ADDRESS) ..... M.D.

.....

.....

Westwood Pharmacal Corp.

1020 MAIN ST., BUFFALO 2, N. Y.

"Pianissimo,"

"Pianissimo," mute command of the maestro . . . with a measure of significance for the syphilologist. In the clinic, as in the concert hall, it is often necessary to call for *milder* action. This is especially true in the treatment of cardiovascular and hepatic syphilis and latent syphilis in elderly persons where intensive, energetic therapy may cause serious reactions. Too vigorous treatment must be avoided, yet undertreatment guarded against. • For such cases Bismarsen offers definite advantages. The spirillicidal effect, although somewhat slower than that of the arsphenamines, is nevertheless pronounced. • Since it is injected *intramuscularly*, Bismarsen is widely used where the intravenous route is inaccessible as in infants, children, and the obese. • Bismarsen is a chemical combination of 13% arsenic and 23% bismuth. It is obtainable in 0.1-Gm. and 0.2-Gm. ampoules with a corresponding number of ampoules of a special solvent (a weak Solution of Butyn Sulfate). ABBOTT LABORATORIES, North Chicago, Ill.

Bismarsen

REG. U. S. PAT. OFF.

(BISMUTH ARSPHENAMINE SULFONATE, D-R-L)

New Tests Show Value of Mild Soap Routine

IN advising women on steps toward skin health and skin beauty, dermatologists have long stressed the importance of proper daily cleansing—with mild soap and water.

To make certain that the skin cleansing routine which we recommend—the Camay Mild-Soap Diet—is correct in every detail, we have consulted dermatologists and incorporated their suggestions.

This method—and Camay—have recently been subjected to careful clinical tests. Competent dermatologists studied the effects on skin of over 100 women who changed from their usual cleansing methods to the Camay Mild-Soap Diet. The results showed that the Camay routine was beneficial to skin in a very high percentage of the cases—that Camay cleanses the skin gently, thoroughly, and without irritation.

Repeated skin tests—patch tests, immersion tests—have demonstrated further that Camay fulfills the most rigid requirements for mildness.

If you are called upon to recommend a toilet soap for daily use, we believe that you may recommend Camay with confidence. For in addition to its outstanding mildness, it lathers richly and has a delicate fragrance particularly appealing to women.

Samples of Camay are available to physicians for personal or office use and observation. Simply address your written request to Procter and Gamble, Cincinnati, Ohio.

CAMAY

A product of Procter and Gamble

NEOARSPHENAMINE:
For intravenous administration in aqueous solution.

SULPHARSPHENAMINE:
For deep intramuscular injection in aqueous solution; preferred for children and other individuals where intravenous administration of an arsenical is difficult. Particularly suitable in congenital syphilis.

DICHLOROPHENARSINE HYDROCHLORIDE: A highly stable, well tolerated and efficacious arsenoxide preparation, given intravenously.

IODOBISMITOL WITH BENZOCAINE:
A propylene glycol solution containing 6 per cent sodium iodobismuthite, 12 per cent sodium iodide, and 4 per cent benzocaine (local anesthetic), for intramuscular injection in both early and late syphilis.

SOBISMINOL CAPSULES:
A complex organic anionic bismuth compound resulting from the interaction of sodium bismuthate, triisopropanolamine, and propylene glycol; for oral administration; also in ampuls for intramuscular use.

BISMUTH SUBSALICYLATE IN OIL: Widely used as a source of the heavy metal, bismuth; given intramuscularly.

TRYPARSAMIDE: Freely soluble in water; used intravenously; indicated specifically in treatment of neurosyphilis.

E·R·SQUIBB & SONS

Manufacturing Chemists to the Medical Profession Since 1858

Archives of Dermatology and Syphilology

VOLUME 49 JUNE 1944 NUMBER 6

COPYRIGHT, 1944, BY THE AMERICAN MEDICAL ASSOCIATION

KERATOSIS BLENNORRHAGICA WITHOUT GONORRHEA (REITER'S DISEASE?)

WALTER F. LEVER, M.D., AND G. MARSHALL CRAWFORD, M.D.

BOSTON

Recently 2 patients were observed at the Massachusetts General Hospital whose cutaneous lesions suggested a gonorrheal eruption but for whom bacteriologic studies excluded the presence of a gonococcic infection. The first had cutaneous lesions identical with keratosis blennorrhagica and, in addition, chronic arthritis, urethritis and conjunctivitis. The second had cutaneous lesions which were primarily purpuric and vesicular. On some of the lesions, particularly those situated over bony prominences, thick, hyperkeratotic scales developed, and they then resembled keratosis blennorrhagica. Chronic arthritis and conjunctivitis were present, but there was no urethritis.

On the basis of the cases of these 2 patients and of cases described in the literature it will be stressed that lesions identical with those of keratosis blennorrhagica may occur in the absence of a gonococcic infection. The diagnosis of Reiter's disease has been suggested by some authors for lesions of this type.

RELATIONSHIP BETWEEN KERATOSIS BLENNORRHAGICA AND GONORRHEA

Although keratosis blennorrhagica is regarded by most authors as a specific gonorrheal lesion, recent reports indicate that it may also occur in patients who are free from gonococcic infection. Prior to the introduction of cultural methods for the diagnosis of gonorrhea, it was frequently difficult to ascertain or to exclude a gonococcic infection. This fact may explain why the opinion that keratosis blennorrhagica may occur in patients without gonorrhea has gained ground only slowly. Buschke,[1] in 1912, was the first to express this opinion, but in 1923 [2] he abandoned this concept and concluded that in all cases keratosis blennorrhagica develops on the basis of gonorrhea. When, however, in 1928 he encountered another case of this disease in which there was no evidence of gonorrhea,[3] Buschke reverted to the opinion which he had previously expressed in 1912. Some authors, like Waelsch,[4] Falk [5] and Adamson,[6] regarded cases of keratosis blennorrhagica in which a gonococcic infection could be excluded as cases of pustular psoriasis, a point of view which has been opposed by Buschke [3] and Rostenberg and Silver.[7]

Additional cases of keratosis blennorrhagica in which a gonorrheal infection could be definitely excluded have been reported in recent years by Rostenberg and Silver,[7] Lojander,[8] Löhe and Rosenfeld,[9] Wiedemann,[10] Kuske [11] and Kruspe [12] (case 6). Rostenberg and Silver suggested the name "dermatitis rupioides arthropathica" in place of keratosis blennorrhagica, in order to denote that in not all cases was it associated with gonorrhea. Wiedemann, Kuske and Kruspe gave to their cases the designation of Reiter's disease. Kuske even went so far as to

From the Dermatologic Department of the Massachusetts General Hospital, C. Guy Lane, Chief.

1. Buschke, A.: Ueber universell symmetrische entzündliche Hyperkeratosen auf uroseptischer und arthritischer Basis, Arch. f. Dermat. u. Syph. **113**:223, 1912.

2. Buschke, A., and Langer, E.: Hyperkeratotische Exantheme bei Gonorrhöe und ihre Beziehungen zur Psoriasis, Dermat. Wchnschr. **76**:145, 1923.

3. Buschke, A.: Ueber die sog. "Gonorrhoischen Hyperkeratosen" ohne Gonorrhöe, Klin. Wchnschr. **7**: 1133, 1928.

4. Waelsch, L.: Ueber die Beziehungen zwischen Psoriasis und Gelenkserkrankungen, Arch. f. Dermat. u. Syph. **104**:195 and 453, 1910.

5. Falk, A.: Psoriasis arthropathica (einschliesslich der sog. "hyperkeratotischen Exantheme" bei gonorrhoischen Gelenkerkrankungen), Arch. f. Dermat. u. Syph. **129**: 299, 1921.

6. Adamson, H. G.: Keratodermia Blennorhagica: Is It a Form of Psoriasis? Brit. J. Dermat. **32**: 183, 1920.

7. Rostenberg, A., and Silver, H.: Keratoderma Blennorrhagicum, Arch. Dermat. & Syph. **16**:741 (Dec.) 1927.

8. Lojander, W.: Ueber Keratodermien im Anschluss an gelenkaffektionen gonorrhoischen und nichtgonorrhoischen Ursprungs, Acta dermat.-venereol. **8**:227, 1927.

9. Löhe, H., and Rosenfeld, H.: Klinische und physiologisch-chemische Untersuchungen über die Hyperkeratosenbildung bei Gonorrhöe und bei Psoriasis pustulosa arthropathica, Dermat. Ztschr. **55**:355, 1929.

10. Wiedemann, H.: Reitersche Erkrankung, Wien. klin. Wchnschr. **47**:1245, 1934.

11. Kuske, H.: Ueber die Hauterscheinungen bei Morbus Reiter, Arch. f. Dermat. u. Syph. **179**:58, 1939.

12. Kruspe: Zur Aetiologie der Reiterschen Erkrankung, Dermat. Wchnschr. **112**:457, 1941.

question the gonorrheal causation in all cases of keratosis blennorrhagica and expressed the belief that it was always a manifestation of Reiter's disease. This point of view seems, however, not supported by facts.

REITER'S DISEASE

The term "Reiter's disease" has been used in recent years to designate a clinical syndrome consisting of arthritis, conjunctivitis and urethritis not caused by gonococci or other known micro-organisms. Since 1916, when Reiter [13] reported the first case, 45 cases have been reported in twenty-four communications.[14] It is doubtful, however, whether in all these cases the patients actually had Reiter's disease. In some the triad was not complete; in others the bacteriologic studies were not sufficient to exclude the presence of a gonococcic infection. All but 1 of the reports have come from European countries. The first American report was published recently by Bauer and Engleman.[14t] Bauer and co-workers have since described 5 additional cases (to be published).

In all cases reported so far Reiter's disease has occurred in young adult males. Nearly half (22 patients) were soldiers on active duty in the First or the Second World War. In most cases the arthritis dominated the clinical picture, usually involving several joints, though at times it was monoarticular. Clinically, the arthritis resembled gonorrheal arthritis in many respects. It persisted for periods varying from one to five months, during which time there might be remissions followed by exacerbations. Permanent damage to the joints has not been reported. The urethritis and conjunctivitis were usually of considerably shorter duration than the arthritis and subsided after a few weeks, though they might recur in the course of the disease. The conjunctivitis at times was complicated by iritis or keratitis. Recurrences of the disease might occur months and even years after the original attack has subsided.

Cutaneous lesions have been reported in 9 of the 45 cases. In 6 cases, reported by Wiedemann,[10] Kuske [11] (cases 1 to 4) and Kruspe [12] (case 6), the lesions had the appearance of keratosis blennorrhagica. In 3 cases, reported by Beck,[14n] Postma [14p] and Bauer and Engleman,[14t] the cutaneous lesions were limited to the glans penis and presented the appearance of balanitis circinata.

On a clinical basis alone, a differentiation of Reiter's disease from the gonorrheal syndrome composed of arthritis, conjunctivitis and urethritis is usually impossible. It seems, however, that in Reiter's disease the manifestations usually are less severe: There is less fever and no chills, the disease is self limited in its individual attacks and complete restitution of articular function is the rule. Furthermore, lack of response to sulfonamide compounds is in favor of Reiter's disease. Nevertheless, the only reliable method of distinction between the two diseases is by thorough bacteriologic studies, since in Reiter's disease smears and cultures of material obtained from the urethra, prostate, cervix, joint fluid and cutaneous lesions are always negative for gonococci. The value of the gonococcus complement fixation test in the differentiation of the two diseases is limited. In Reiter's disease the reaction should be negative, unless the patient previously had gonorrhea. In gonococcic arthritis, on the other hand, the gonococcus complement fixation

13. Reiter, H.: Ueber eine bisher unbekannte Spirochäteninfektion (Spirochaetosis arthritica), Deutsche med. Wchnschr. **42**:1529, 1916.

14. (*a*) Fleischmann, in discussion on Reiter.[13] (*b*) Michael, M.: Urethritis non gonorrhoica: Arthritis pseudogonorrhoica, Dermat. Ztschr. **24**:419, 1917. (*c*) Sommer, A.: Drei wahrscheinlich als "Spirochaetosis arthritica" (Reiter) anzusprechende Krankheitsfälle, Deutsche med. Wchnschr. **44**:403, 1918. (*d*) Junghanns, O.: Ein weiterer Fall von Urethritis non gonorrhoica und septischer Allgemeinerkrankung, ibid. **44**: 1304, 1918. (*e*) Stühmer, A.: Ueber die sogenannte "Spirochaetosis arthritica" (Reiter), München. med. Wchnschr. **68**:769, 1921. (*f*) Frühwald, R.: Beitrag zur sogenannten Spirochaetosis arthritica (Reiter), Dermat. Ztschr. **51**: 35, 1927; So-Called Spirochaetosis Arthritica (Reiter, 1916), Urol. & Cutan. Rev. **32**:7, 1928. (*g*) Paetzel, H.: Ueber Polyarthritis nach unspezifischer Urethritis, Med. Klin. **24**: 134, 1928. (*h*) Freund, E.: Gelenkerkrankungen, Berlin, Urban & Schwarzenberg, 1929, p. 144. (*i*) Kristjanssen, A.: Fall zur Diagnosis, Zentralbl. f. Haut- u. Geschlechtskr. **31**:165, 1929-1930; Nichtgonorrhoische Urethritis mit Conjunctivitis und Arthritis, ibid. **34**:855, 1930. (*j*) Foerster, R.: Zwei Fälle von Reiterscher Erkrankung, Med. Welt **7**:700, 1933. (*k*) Balban, W.: Zur Kenntnis der sog. "Spirochaetosis arthritica" (Reiter), Dermat. Ztschr. **68**:305, 1934. (*l*) Moltke, O.: Polyarthritis urethritica, Acta med. Scandinav. **89**:696, 1936. (*m*) Pfleger, R.: Zur Aetiologie der Reiter'schen Erkrankung, Med. Klin. **33**:465, 1937. (*n*) Beck, F.: Ein Beitrag zur der sog. Spirochaetosis arthritica Reiter, ibid. **33**:1162, 1937. (*o*) Thesleff, C.: Ett fall av urethritis nongonorrhoica med led- och ögon komplikationer, Finska läk.-sällsk.handl. **80**:411, 1937. (*p*) Postma, C.: A Case of Reiter's Disease, Acta dermat.-venereol. **18**:691, 1937. (*q*) Clemmesen, S., and Kalbak, K.: Polyarthritis urethritica simplex hos. 4 patienter, Ugesk. f. læger **100**:618, 1938. (*r*) Usseglio, G., and Zancan, B.: Il morbo di Reiter, Arch. per le sc. med. **69**:79, 1940. (*s*) Kardung: Ein unter dem Bilde der Reiterschen Erkrankung verlaufender Fall von Urethritis, Arthritis und Conjunctivitis, Med. Welt **15**:403, 1941. (*t*) Bauer, W., and Engleman, E. P.: A Syndrome of Unknown Etiology Characterized by Urethritis, Conjunctivitis and Arthritis (So-Called Reiter's Disease), Tr. A. Am. Physicians **57**:307, 1942. (*u*) Wiedemann.[10] (*v*) Kuske.[11] (*w*) Kruspe.[12] (*x*) Reiter.[13]

test usually elicits a positive reaction—according to Bauer, Ropes and Short [15] in at least 85 per cent of cases. The fact, however, that false positive reactions occur not infrequently should be kept in mind. (Because of its unreliability no laboratory in Boston performs this test any more.)

The cause of Reiter's disease is not known, and no successful treatment is available. Sulfonamide compounds have proved ineffective. Karsting [16] expressed the belief that by fever therapy he had been able to prevent recurrence in his patient, who already had had more than ten attacks.

REPORT OF CASES

Case 1.—A 37 year old white man noted in March 1936 a glairy mucoid discharge from the urethra accompanied by urinary frequency and dysuria. The urethral discharge disappeared after one week. At that time the patient began to note pain and swelling of the knees and ankles. In the course of the next two weeks the joints of the hands, feet, elbows and spine became involved. About one month after onset of the illness, cutaneous lesions appeared which the patient described as resembling "bits of soft cement." The eruption involved the back and the nail beds and nail folds of the fingers. Simultaneously, a bilateral conjunctivitis and iritis developed. The patient spent four months in a hospital. Gonococci were not found in smears from the urethra. The arthritis, cutaneous eruption and conjunctivitis persisted for four months and then disappeared completely. From July 1936 to May 1942 the patient enjoyed good health.

In May 1942 he began to note aching in many joints, particularly in those of the knees, ankles, hands, wrists and spine. At the same time there was a recurrence of the glairy mucoid discharge from the urethra. He stated that he had had no recent sexual exposure. The urethral discharge persisted for only a few days. The arthritis, however, gradually became more severe. In July 1942 the patient noted the appearance of small whitish "blisters" between the fingers. The lesions grew to about the size of a small pea and became covered with thick scales. The eruption soon spread to involve the skin of the hands, feet, ankles, back and face. In August 1942 the patient was admitted to a Boston hospital. Smears and cultures of material from the urethra and prostate did not reveal gonococci, and a gonococcus complement fixation test elicited a negative reaction. He was given a course of sulfathiazole, but the arthritis became more severe and the cutaneous eruption continued to spread. On September 28, the patient was transferred to the dermatologic ward of the Massachusetts General Hospital.

Physical Examination.—On admission the patient appeared severely ill. He was cachectic and almost unable to move because of severe pains in the joints.

Skin: Lesions were present over the entire body, but the hands, feet, face and back showed the most severe involvement. The earliest lesions were flat-topped flaccid pustules which varied in diameter from 0.2 to 2 cm. and were surrounded by erythema. They contained no fluid but only small amounts of macerated material. Older lesions showed heaped-up, hard, horny scales which were adherent to their base (fig. 1 *A*). By peripheral extension and coalescence large hyperkeratotic lesions had formed on the hands, feet and face. On the right sole a large ulcer covered with thick, brownish, keratotic scales was present (fig. 1 *B*). The fourth and fifth toes of the right foot were edematous, and their skin was covered with thick adherent scales (fig. 1 *C*). The face presented a large area of involvement on the nose and cheeks of "butterfly distribution." The border of this lesion was raised, erythematous and studded with small pustules, while the center was covered with greenish brown keratotic scales. The nails of several fingers and toes were lifted from their beds by accumulations of keratotic scales. Some of the nails showed disintegration at the lateral borders and the free margin.

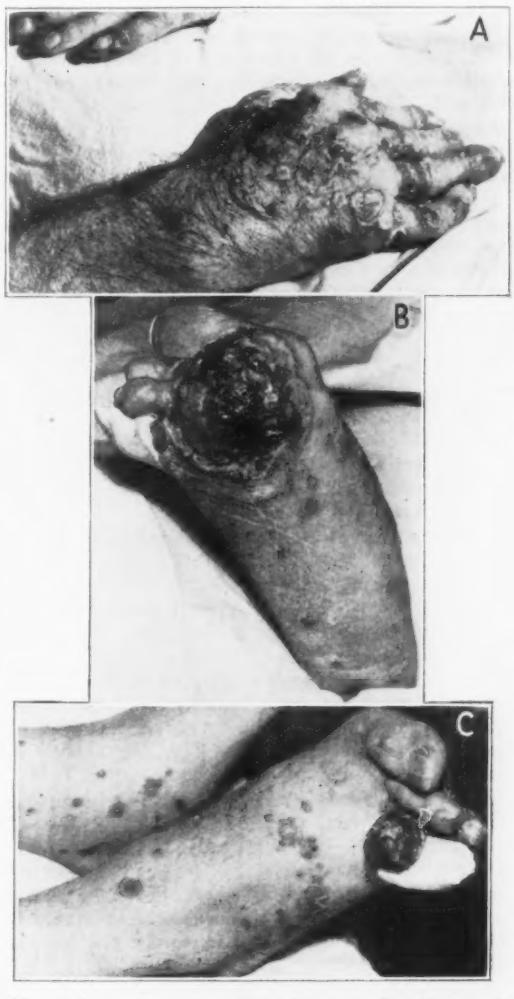


Fig. 1 (case 1).—*A*, early lesions are flaccid pustules containing no fluid but only macerated material. Older lesions are hyperkeratotic. Several of the fingers show arthritic deformities. *B*, a large ulcer covered with thick, brownish, keratotic scales on the right sole. *C*, pustular and hyperkeratotic lesions on the feet. The fourth toe of the right foot is considerably swollen and covered with thick, adherent scales.

Skeletal System: There was severe pain with limitation of motion in most joints, particularly in the small joints of the hands and feet and in the wrists, knees, ankles, elbows and spine. The joints of the

15. Bauer, W.; Ropes, M. W., and Short, C. L.: The Treatment of the Infectious Arthritides with Sulfonamide Compounds, M. Clin. North America **26**:1529, 1942.

fingers and toes showed swelling and subluxation. The wrists were swollen, and there was ulnar deviation of both hands. The knees and ankles exhibited slight symmetric swelling with effusion into both knee joints.

Genitourinary System: The glans penis showed circumscribed areas of erythema and scaling. There was no urethral discharge. The prostate and seminal vesicles felt normal on palpation. The prostatic fluid, however, contained many pus cells.

Eyes: The conjunctivas were moderately injected. A small amount of purulent exudate was present in the conjunctival sacs. The scleras and iris appeared normal.

Course of the Disease.—Beginning on the fifth day of hospitalization, the patient was given sulfathiazole, 6 Gm. a day. On the fourth day of this treatment the sulfathiazole level in the blood was 3.8 mg. per hundred cubic centimeters and on the seventh day 4.3 mg. No essential change in the general condition was noted. The temperature was normal or nearly normal in the morning, but it rose to 101 or 102 F. in the evening. On the sixteenth day in the hospital the patient suddenly complained of a feeling of substernal oppression. The respiration became rapid and shallow, cyanosis developed and the blood pressure fell to 55 systolic and 30 diastolic. An electrocardiogram was suggestive of recent anterior myocardial infarct, in contrast to one taken six days previously, which had been normal. The patient died two days later. Permission for an autopsy was not obtained.

Laboratory Data.—The urine was essentially normal. Erythrocyte counts varied from 3,800,000 to 4,500,000 and leukocyte counts from 6,000 to 31,000. Differential counts were normal. The result of a serologic test for syphilis (Hinton) was negative.

Blood Chemistry: Uric acid measured 2.1 mg., total protein 6.1 and 5.5 Gm., nonprotein nitrogen 18 mg., blood sugar 97 mg., calcium 9.3 and 9.5 mg. and phosphorus 5.0 and 3.6 mg. per hundred cubic centimeters. The phosphatase activity was 7.3 units.

Roentgen Examination: Roentgenograms of the hands and wrists showed atrophy about all the visible joints. The knees showed considerable flecky atrophy with narrowing of the left joint space. Similar changes were observed in the spine. The diagnosis of the roentgenologist was active rheumatoid arthritis.

Biopsy: A biopsy specimen from an early cutaneous lesion showed several small intraepithelial vesicles filled with degenerated epithelial cells, polymorphonuclear leukocytes and lymphocytes. The epithelium around the vesicles showed edema, moderate acanthosis and infiltration with polymorphonuclear leukocytes. The horny layer was thickened, parakeratotic and infiltrated with polymorphonuclear leukocytes. The corium in its papillary and subpapillary layers showed dense infiltration with polymorphonuclear leukocytes and lymphocytes. The histologic picture was regarded as consistent with keratosis blennorrhagica in its early "pustular" stage.

Synovial Fluid Count: An examination of synovial fluid obtained from the left knee joint revealed slightly bloody fluid. The erythrocyte count was 510,000 and the leukocyte count 6,900 per cubic millimeter. The differential count showed 93 per cent neutrophils, 4 per cent monocytes and 3 per cent lymphocytes.

Bacteriologic Studies (performed by Dr. William E. Smith, Bacteriologic Laboratory of the Massachusetts General Hospital): Direct microscopic examinations, cultures, animal inoculations and immunologic tests failed to demonstrate a specific organism which could have been held responsible for the clinical symptoms. The following studies were made:

Direct Microscopic Examinations: Material from the conjunctivas and the prostate showed a few gram positive organisms, presumably diphtheroids. Giemsa stains showed no inclusion bodies. Microscopic examination of scrapings of skin, and of blood and joint fluid showed no organisms or inclusion bodies. Dark field examinations did not reveal spirochetes.

Cultures: Cultures were made aerobically, anaerobically and under increased carbon dioxide tension on blood agar plates and ascitic agar plates, in rabbit serum broth and in Brewer's thioglycollate broth enriched with ascitic fluid. Cultures from the conjunctivas and the prostate yielded diphtheroids, which were regarded as components of the normal flora. Cultures of blood and joint fluid remained sterile. Cultures of cutaneous lesions either remained sterile or yielded staphylococci. Rabbit serum broth cultures incubated anaerobically were examined for spirochetes by dark field illumination, with negative results. Gonococci were especially sought but were not found. Pleuropneumonia-like (L) organisms whose association with genitourinary infections has been described[16] were also especially sought, but with negative results.

Animal Inoculations: Pus from the left conjunctiva was introduced onto the scarified cornea and into the anterior chamber of the eye of a rabbit. An emulsion of cutaneous scrapings was injected into rabbits intravenously and intraurethrally and was rubbed into the scarified skin of a rabbit. Synovial fluid from the left knee was injected into foot pads of mice and into the knee joint of a rabbit. Lesions were not produced.

Immunologic Tests: The result of a gonococcus complement fixation test was negative. (Dr. J. F. Mahoney, United States Marine Hospital, Staten Island, N. Y., performed this test.)

Diagnosis: The diagnoses were chronic arthritis (rheumatoid?), urethritis, conjunctivitis, keratosis blennorrhagica; Reiter's disease (?).

Case 2.—A 34 year old married white woman had a mild sore throat of one week's duration early in May 1941. One week after its disappearance she first noted pain in her left shoulder and elbow. In the course of a few weeks many other joints became affected. Three days after the onset of the arthritis cutaneous lesions began to form, mainly on the extremities but also on the abdomen, buttocks, face and lips. The earliest lesions consisted of purpuric spots and small blisters. The blisters would break and leave open sores which were tender. These healed slowly and some before healing became covered with thick, dark, horny scales. At one time, soon after the onset of the eruption, the patient had noted a few lesions on the mucous membranes of the lips and on the palate. Late in June 1941 she had bilateral conjunctivitis with purulent discharge for five days. Sensitivity to light persisted for four weeks. She had several mild recurrences of the conjunctivitis after this. Also late in June she had two severe attacks of abdominal cramps, both were followed by some blood in the stool. The patient never noted urethral or vaginal discharges or symptoms.

The arthritic pains and the cutaneous eruption persisted up to the time of her admission to the hospital, a period of eighteen months. The arthritis had been

16. Dienes, L., and Smith, W. E.: Relationship of Pleuropneumonia-like (L) Organisms to Infections of Human Genital Tract, Proc. Soc. Exper. Biol. & Med. **50**:99, 1942.

most severe during the first few months of the disease; then it began to improve intermittently. The eruption had been severe at the onset and then became less widespread although at times there were exacerbations. During the three months preceding her admission the number of new lesions had again increased. On Nov. 28, 1942, the patient was admitted to the dermatologic ward of the Massachusetts General Hospital.

Physical Examination.—On admission the patient appeared pale and poorly nourished. She did not feel ill but complained of considerable tenderness of the cutaneous lesions. Motion of the hands and feet produced mild pain in the joints.

Skin: Cutaneous lesions were most numerous on the face and extremities. Only a few scattered lesions were noted on the trunk. On the face and extremities lesions were present largely over pressure areas, as on the cartilaginous prominences of the ear and on the cheeks, elbows, extensor surfaces of the arms, wrists, knuckles, tips of the fingers, buttocks, posterior surfaces of the thighs, knees, shins, ankles, heels, balls of the feet and tips of the toes. The lesions were discrete and round, varying in diameter from 1 mm. to 1 cm. The primary lesions were either hemorrhagic puncta or vesicles, many of which had a hemorrhagic base (fig. 2 *A*). At the site of broken vesicles there were erosions or superficial ulcerations. Some lesions were covered with hemorrhagic crusts; others, particularly those located over the bony prominences of the elbows, knees, knuckles, balls of the feet and tips of the fingers and toes, showed thick, dark, heaped-up, horny scales (fig. 2 *B* and *C*). Some healed without scarring; others left superficial atrophic pigmented scars. Scarring was most severe on the shins (fig. 2 *D*).

Mucous Membranes: The hard palate showed at the time of admission seven pinpoint-sized vesicles or erosions. After one week the oral lesions had disappeared, and new ones were not seen subsequently.

Skeletal System: The elbows, knees, wrists and ankles and the joints of the hands and feet were affected. Most severely involved were the joints of the fingers, which showed swelling, ankylosis and deformities to a moderate degree. The metacarpophalangeal joints were flexed and the interphalangeal joints hyperextended. There were moderate limitation of motion of the wrists and of the right elbow and slight limitation of motion of both knees and ankles and of the joints of the toes.

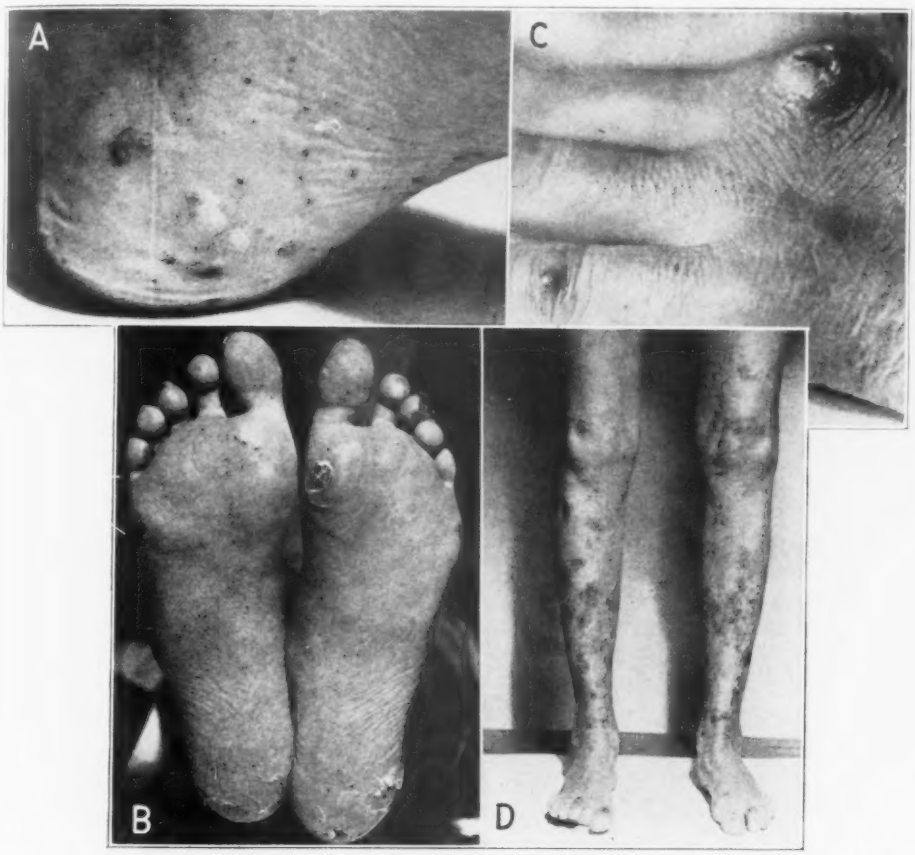


Fig. 2 (case 2).—*A*, on the left heel are purpuric lesions and three vesicles. Two of the vesicles have a hemorrhagic base. *B*, hyperkeratotic lesions resembling keratosis blennorrhagica on the tips of both third toes, the lateral ball of the right foot and medial ball of the left foot. *C*, an early vesicular lesion on a hemorrhagic base on the little finger of the left hand and a hyperkeratotic lesion over the third metacarpal-phalangeal joint. *D*, superficial atrophic pigmented scars, the end stage in the development of the vesicular lesions.

Course of the Disease.—During the first five weeks of the patient's stay in the hospital tenderness of the joints completely disappeared and motion of the joints improved. The skin, however, did not improve, and

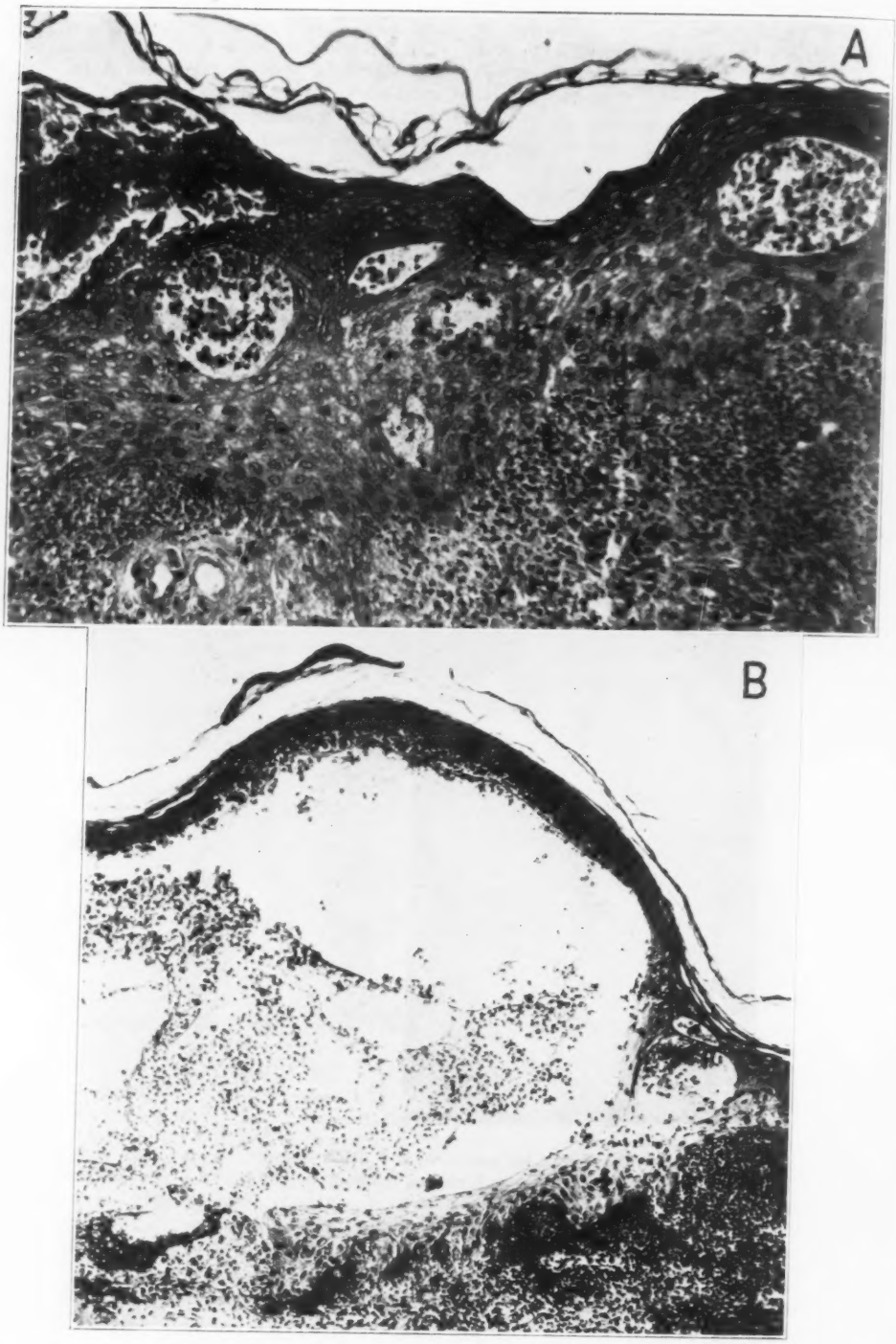


Fig. 3 (case 2).—*A*, section of an early lesion. There are several small intraepidermal vesicles filled with degenerated epidermal cells, erythrocytes, polymorphonuclear leukocytes and lymphocytes. *B*, section of a well developed vesicle. The vesicle is traversed by a network of fibrin and contains degenerated epidermal cells, polymorphonuclear leukocytes and lymphocytes.

frequent new crops of purpuric lesions and vesicles appeared. During the sixth week of hospitalization treatment with sulfadiazine (4 Gm. a day) was begun. After two days the sulfadiazine level in the blood was 6.4 mg. per hundred cubic centimeters. After seven days use of the drug was discontinued because sulfadiazine crystals were found in the urine. New crops of lesions continued to appear. During the eighth week of hospitalization a series of intravenous injections of typhoid organisms was begun. In the course of five weeks fifteen injections were given. The first injection contained 10,000,000 organisms; the last, 1,000,000,000 organisms. Most of the injections were followed by a rise of temperature to 103 and 104 F. Three weeks after the first injection, at a time when seven injections had been given, the number of new lesions had greatly diminished. During the fourth week no new lesions appeared. During the fifth week only two small vesicles formed. Four days after the last injection, on Feb. 23, 1943, the patient was discharged. At the time of her discharge all lesions had either healed or had well progressed toward healing.

Laboratory Data.—The urine and blood cell counts were essentially normal. The sedimentation rate was moderately increased. A tourniquet test elicited a negative reaction. A tuberculin test (old tuberculin in a dilution of 1:10,000) produced slight induration after forty-eight hours. The result of a serologic test for syphilis (Hinton) was negative.

Blood Chemistry: Uric acid measured 2.1 mg., total protein 5.1 Gm., ascorbic acid 0.6 mg., calcium 9.5 mg. and phosphorus 2.8 mg. per hundred cubic centimeters. The phosphatase activity was 3.5 units. On Dec. 2 and Dec. 6, 1942, there was a trace of bromide in the blood.

Roentgen Examination: A roentgenogram of the chest showed old, scarred, apical tuberculosis. Roentgenograms of the hands showed many of the interphalangeal joints narrowed and irregular. There was considerable decalcification in the bones about all joints. The conditions observed were regarded as consistent with rheumatoid arthritis.

Biopsy: Three specimens for biopsy were taken, one including an early vesicular lesion, one including a well developed vesicle and one from an atrophic area. The microscopic picture of the early lesion (fig. 3*A*) closely resembled that of the section obtained from the first patient and appeared consistent with keratosis blennorrhagica in its early "pustular" stage. The section including a well developed vesicle (fig. 3*B*) showed the vesicle to contain degenerated epithelial cells, polymorphonuclear leukocytes, lymphocytes and a network of fibrin. The surrounding epithelium and the underlying corium showed dense infiltration with polymorphonuclear leukocytes and lymphocytes. The section from an atrophic area showed thinning of the epithelium and dense fibrosis of the corium.

Bacteriologic Studies (performed by Dr. William E. Smith).—All direct microscopic examinations, cultures and animal inoculations gave essentially negative results.

Urethral and cervical smears and material from the cutaneous lesions revealed no gonococci, inclusion bodies or spirochetes. Urethral and cervical cultures were repeatedly negative for gonococci and pleuropneumonia-like (L) organisms. Fluid from vesicles and tissue of a skin lesion were inoculated into the mediums described in the first case and also into Loewenthal's medium for the growth of tubercle bacilli, into Maitland tissue culture flasks containing chick embryo tissue suspended in Tyrode's solution, and into developing chick embryos. Fluid from vesicles was inoculated into the skin, testis, cornea and anterior chamber of the eye of rabbits. The results were essentially negative.

Immunologic Tests: Two gonococcus complement fixation tests were performed. The result of one was reported as positive and that of the other as negative. (Dr. J. F. Mahoney performed these tests.) Agglutination reactions with typhoid bacilli and the Flexner, Shiga and Sonne strains of dysentery bacilli were negative. Agglutination reactions with the Hiss strain of dysentery bacilli were positive with a dilution of 1:40, but this was not considered significant.

Diagnosis.—The diagnoses were chronic arthritis (rheumatoid?), conjunctivitis, purpuric and vesicular eruption with subsequent formation of lesions resembling keratosis blennorrhagica in some areas; Reiter's disease(?).

Follow-Up Studies: Within three weeks after the patient returned home new lesions began to form on her skin. When she was last heard of, in August 1943, the cutaneous eruption was as severe as it had been at the time of her admission to the Massachusetts General Hospital. Treatment with large doses of vitamin A and diethylstilbestrol had no effect on the cutaneous eruption.

COMMENT

No definite diagnosis seems possible in either case.

In case 1 the disease of the joints closely resembled rheumatoid arthritis, while the cutaneous lesions presented the appearance of keratosis blennorrhagica. Thorough and repeated bacteriologic studies failed to show gonococci, and the results of 2 gonococcus complement fixation tests were negative. The presence of lesions of the type of keratosis blennorrhagica in the absence of gonorrhea represents the most important finding in this case. The association of arthritis, urethritis and conjunctivitis in the absence of a gonococcic infection necessitates consideration of Reiter's disease. The presence of lesions of the type of keratosis blennorrhagica was regarded as consistent with a diagnosis of Reiter's disease, since it has been reported in 6 of the 45 cases in the literature. However, the articular involvement was unlike any reported in cases of this disease. In all cases of Reiter's disease reported so far, the arthritis has never been progressive but has lasted only a few months and left no permanent damage. In this case the first attack of arthritis resulted in complete recovery, but the second attack, six years later, was progressive and caused considerable destruction of the joints. The fact that the disease caused the death of the patient is also unusual, since in none of the reported cases of Reiter's disease did the disease cause death. Because it is believed that in the present stage of knowledge about Reiter's disease only cases that

correspond to the syndrome in all respects should be included, we hesitate to attach the diagnosis of Reiter's disease to this case.

In case 2 the arthritis, as in the previous case, resembled rheumatoid arthritis closely. There was a moderate amount of articular destruction. The cutaneous eruption showed as primary lesions either hemorrhagic puncta or vesicles. Some of the lesions later assumed the appearance of keratosis blennorrhagica. On one occasion small vesicles and erosions were noted on the hard palate. There had been several attacks of conjunctivitis. It was regarded as significant that the cutaneous eruption had appeared simultaneously with the arthritis and had persisted, as had the arthritis, up to the time of the patient's admission to the hospital, a period of eighteen months. This was thought to indicate a close relation between the two clinical manifestations.

On the basis of the clinical appearance, the possibility of gonorrheal eruption and gonorrheal arthritis was considered. Hemorrhagic and vesicular eruptions have been described in the literature as gonorrheal eruptions, occurring with or without lesions of keratosis blennorrhagica. Buschke,[17] in his classification of gonorrheal eruptions, distinguished four types: first, simple erythemas; second, urticarial and erythema-nodosum-like eruptions; third, hemorrhagic and vesicular eruptions, and fourth, hyperkeratotic lesions. In a later paper [1] he stated that a sharp separation of these four types of gonorrheal eruptions is not always possible and that various combinations of the four types may occur. The number of cases reported in which there were hemorrhagic and vesicular eruptions is small. Hauck[18] in 1935 counted approximately 30 cases of "purpura gonorrhoica" in the literature. Combined vesicular and hemorrhagic eruptions have been described by Hodara,[19] Welander,[20] Dorner[21] and Siegel.[22] The presence of oral lesions in our patient would also be consistent with a diagnosis of gonorrheal eruption, since oral lesions have been reported, though rarely, as occurring in persons with keratosis blennorrhagia. Cases with oral lesions have been reported by Stanislawski,[23] Frei,[24] Guerrieri,[25] Bogrow,[26] Berman,[27] Chambers and Koetter,[28] Epstein,[29] Epstein and Chambers[30] and Sherman.[31] In most of the reported cases the oral lesions consisted of small papules. In Chambers and Koetter's case the surface of the papules became eroded. In Bogrow's case the oral lesions were like the lesions encountered in this case, namely, small vesicles and erosions located on the hard palate.

The impression of a gonorrheal eruption was, however, not supported by laboratory data. Gonococci were not demonstrable in material from the urethra, cervix or cutaneous lesions despite careful search by smears and cultures. Of two gonococcus complement fixation tests, one elicited a positive reaction and the other a negative. The not infrequent occurrence of false positive gonococcus complement fixation reactions makes it impossible to accept one positive result as conclusive evidence of a gonococcic infection. The presence of arthritis and conjunctivitis in the absence of conclusive evidence of a gonococcic infection would suggest, as in the first case, the possibility of Reiter's disease. However, the presence of permanent damage to the joints and the absence of a history of urethritis are points against this diagnosis. The

17. Buschke, A.: Ueber Exantheme bei Gonorrhöe, Arch. f. Dermat. u. Syph. **48**:181 and 385, 1899.

18. Hauck, L.: Enantheme und Exantheme beim Tripper, in Arzt, L., and Zieler, K.: Die Haut- und Geschlechtskrankheiten, Berlin, Urban & Schwarzenberg, 1935, vol. 5, p. 380.

19. Hodara, M.; Osman Bey; Izet Bey, and Chevkiet Bey: Ein Fall von Gonokokkämie und generalisiertem gonorrhoischem Exanthem, Dermat. Wchnschr. **55**:397, 1912.

20. Welander, E.: Ein Fall von Blennorrhöe, mit Endocarditis und kutanen Manifestationen kompliziert, Monatsh. f. prakt. Dermat. **21**:50, 1895.

21. Dorner, L.: Ueber Gonokokkensepsis, Deutsche med. Wchnschr. **49**:1549, 1923.

22. Siegel, L. A.: A Case of Gonococcus Septicemia, Bull. Buffalo Gen. Hosp. **3**:66, 1925.

23. Stanislawski, W.: Ueber einen Fall von gonorrhoischer Urethritis mit Affektion der Gelenke, symmetrischem hornartigen Hautausschlag und Ausfallen der Nägel, Monatsb. d. Krankh. d. Harn- u. Sex. Appar. **5**:643, 1900.

24. Frei, W.: Gonorrhoisches Exanthem und Enanthem bei Arthritis gonorrhoica, Zentralbl. f. Haut- u. Geschlechtskr. **4**:323, 1922.

25. Guerrieri, T.: Blenoderma cheratosico, Gior. ital. d. mal. ven. **65**:361, 1924.

26. Bogrow, S. L.: Ueber gonorrhoische Keratosen. Ein Fall von gonorroischen Keratosen der Haut und Mundschleimhaut, Arch. f. Dermat. u. Syph. **143**:23, 1923.

27. Berman, L.: Ueber einen Fall von gonorrhoischer Keratose der Haut und Mundschleimhaut, Dermat. Ztschr. **51**:420, 1928.

28. Chambers, S. O., and Koetter, G. F.: Keratosis Blennorrhagica, Arch. Dermat. & Syph. **27**:411 (March) 1933.

29. Epstein, E.: Hyperpyrexia in the Treatment of Keratoderma Blennorrhagicum, Am. J. Syph., Gonor. & Ven. Dis. **21**:148, 1937.

30. Epstein, E., and Chambers, S. O.: Keratosis Blennorrhagica with Corneal Lesions: Further Observations on the Therapeutic Effect of Hyperpyrexia, Arch. Dermat. & Syph. **36**:1044 (Nov.) 1937.

31. Sherman, W. L.; Blumenthal, F., and Heidenreich, J.: Blennorrhagic Balanitiform Keratoderma, Arch. Dermat. & Syph. **39**:422 (March) 1939.

cutaneous eruption also differed from the type of eruptions reported in Reiter's disease. We therefore hesitate, even more than in the first case, to make a diagnosis of Reiter's disease.

SUMMARY

Two patients had cutaneous lesions suggestive of a gonorrheal eruption but without bacteriologic evidence of gonococcic infection. The first patient had arthritis, conjunctivitis, urethritis and cutaneous lesions identical with keratosis blennorrhagica. The second patient had arthritis, conjunctivitis and a cutaneous eruption, the primary lesions of which were hemorrhagic puncta and vesicles. On some of the lesions thick, heaped-up horny scales developed, and they then resembled keratosis blennorrhagica. Urethritis was not present.

No evidence of gonococcic infection could be found in the first case. In the second case, one of two gonococcus complement fixation tests performed elicited a positive reaction, but this was not regarded as conclusive evidence of a gonococcic infection in view of the negative evidence of bacteriologic studies. Organisms which could have been regarded as the cause of the disease were not found in either case.

Reiter's disease was considered as diagnosis for the 2 cases but because of important deviations from the accepted clinical picture of this disease it is doubtful that this term should be employed.

CHROMOBLASTOMYCOSIS

REPORT OF A CASE FROM CONTINENTAL UNITED STATES AND DISCUSSION OF THE CLASSIFICATION OF THE CAUSATIVE FUNGUS

CHAPMAN H. BINFORD, M.D.
Surgeon, United States Public Health Service
NEW ORLEANS
AND
GEORGE HESS, M.D.
Surgeon (R), United States Public Health Service
ATLANTA, GA.
WITH MYCOLOGIC REPORT AND DISCUSSION BY
C. W. EMMONS, PH.D.
Principal Mycologist, United States Public Health Service
WASHINGTON, D. C.

At this time, when there is renewed interest in the study of tropical diseases, it is considered worth while to report another case of chromoblastomycosis occurring in a person who had never been outside the continental United States. It is furthermore considered of value to emphasize the fact that a considerable distance (Atlanta, Ga., to New Orleans) between the physician treating the patient and the laboratory offered no real difficulty in making the diagnosis and culturing the causative fungus.

In the mycologic section of this paper we have corrected the name of the causative fungus to conform with present knowledge of the etiologic agents of this mycosis. The generally accepted name of the disease, chromoblastomycosis, however, has been retained. The substitute term, chromomycosis, which has been proposed is somewhat shorter, but in our opinion it is unsuitable for reasons which have been discussed elsewhere.[1] Many of the recently reported cases illustrate the clinical resemblances which may exist between chromoblastomycosis and American blastomycosis. We believe it to be desirable that the name suggest this similarity. The name has never caused any confusion so far as we are aware, and we see no reason to anticipate that it will do so.

Chromoblastomycosis is actually a cosmopolitan disease, but it has been observed most frequently in tropical or subtropical zones, notably in Brazil, Puerto Rico and Cuba. Nine cases have been previously reported from the continental United States. In view of the excellent reports on the subject which have appeared recently, it is not the purpose of this paper to discuss the general features of the disease. An article by Weidman and Rosenthal[2] has adequately covered the present knowledge, reported 1 case from Philadelphia and summarized 12 others with lesions which were considered until recently to be atypical of the disease. Moore and associates[3] have observed 3 cases in St. Louis. In a recent article reporting the last 2 cases seen there the important features of the disease have been again reviewed. In the present case the disease, while discovered in Atlanta, Ga., appears to have originated in Florida. One unpublished case[1] has previously been reported from that state.

A paper by Pardo-Castello[4] and associates described the condition as it occurs in Cuba and summarized the clinical aspects of 31 cases

From the Laboratory, United States Marine Hospital, New Orleans; the Hospital, United States Penitentiary, Atlanta, Ga., and the Division of Infectious Diseases, National Institute of Health, Washington, D. C.

1. Emmons, C. W.; Hailey, H., and Hailey, H.: Chromoblastomycosis: Report of Sixth Case from Continental United States, J. A. M. A. **116**:25 (Jan. 4) 1941.

2. Weidman, F. D., and Rosenthal, L. H.: Chromoblastomycosis: A New and Important Blastomycosis in North America; Report of a Case in Philadelphia, Arch. Dermat. & Syph. **43**:62 (Jan.) 1941.

3. Moore, M., and Mapother, P.: Chromomycosis of the Face: Report of a Case and a Study of the Causative Organism, Phialophora Verrucosa, Arch. Dermat. & Syph. **41**:42 (Jan.) 1940. Moore, M.; Cooper, Z. K., and Weiss, R. S.: Chromomycosis (Chromoblastomycosis): Report of Two Cases, J. A. M. A. **122**:1237 (Aug. 28) 1943.

4. Pardo-Castello, V.; Rio Leon, E., and Trespalacios, F.: Chromoblastomycosis in Cuba, Arch. Dermat. & Syph. **45**:19 (Jan.) 1942.

observed there. These authors have classified their cases into five different types: (1) verrucous, (2) tuberculoid, (3) syphiloid, (4) psoriasiform and (5) cicatricial and elephantiasic. The lesions are more commonly found on the skin of the lower extremities, may be single or multiple, generally grow slowly and are relatively symptomless.

REPORT OF CASE

A. B., a 53 year old white man, was admitted to the hospital of the United States Penitentiary, Atlanta, Ga., on Jan. 13, 1943 for observation and treatment of a cutaneous lesion on the left leg. He stated that he had been a farmer all of his life, had never been outside the continental United States and had always lived in the vicinity of Punta Gorda, Fla. In 1916 he had been ill with typhoid, and in 1935, with generalized arthritis. Otherwise, his personal history was not unusual.

Fig. 1.—Lesion on the skin of the leg.

Approximately fifteen years before, he had noted in the skin of the anterolateral part of the lower left leg a small rough elevated patch about the "size of a pea." This gradually became larger, but it had not shown any inflammatory reaction until a short time before his admission to the hospital.

Physical examination revealed a robust man who weighed 227 pounds (103 Kg.). With the exception of defective vision, examination of the various systems revealed no abnormalities. There was a raised warty lesion on the anterior aspect of the lower third of the left leg. It was about 2 inches (5 cm.) in diameter and elevated about ⅛ inch (0.3 cm.). There was a zone of erythema about ¼ inch (0.6 cm.) in width encircling the lesion. The surface of this area was dry, and some of the seedlike projections broke off easily with slight trauma.

Laboratory examination revealed 5,200,000 erythrocytes, 8,400 leukocytes, a hemoglobin content of 95 per cent, a normal differential count, a negative Wassermann reaction of the blood and normal urine.

The cutaneous lesion was treated with a dressing of liquid petrolatum. The surface became softer and appeared to be more vascular. The tentative diagnosis was epithelioma. On January 20 a specimen for biopsy was removed and the tissue sent to the laboratory of the United States Marine Hospital, New Orleans.

Microscopic study exhibited irregular epithelial hyperplasia. The interpapillary pegs were elongated and bulky, and the epithelial surface was covered with piled-up keratin. The papillary and subpapillary zones of the corium presented a granulomatous reaction. There were proliferating fibroblasts, many small blood vessels and a copious infiltrate of monocytes, plasma cells and a few neutrophils. Also present were many multinucleate giant cells which frequently contained clusters of distorted, thin-walled, rounded bodies slightly

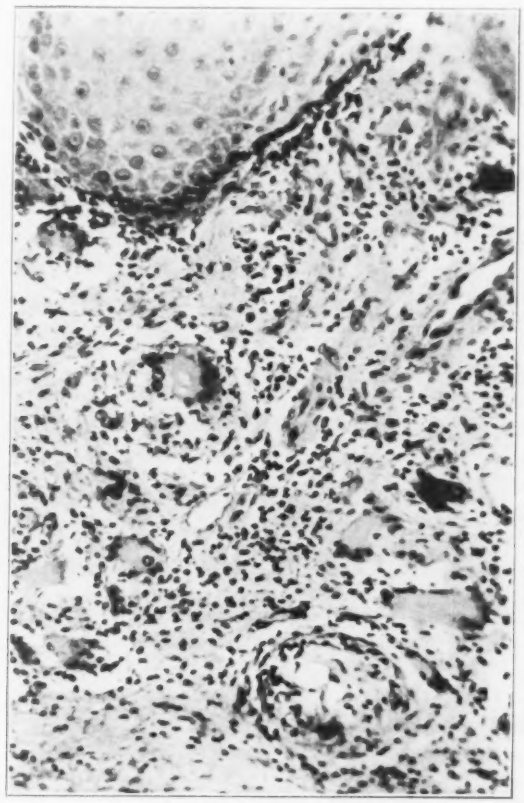


Fig. 2.—Granulomatous reaction in the corium. Note the cells of Phialophora pedrosoi within giant cells, Van Gieson's and Weigert's iron-hematoxylin stain; × 150.

larger than large lymphocytes and without a definite internal structure. Occasionally it appeared that a single wall or septum separated one cell from another. Some of these rounded structures were seen outside the giant cells. They were stained dark green-blue by Giemsa stain, and were light brown in unstained paraffin sections. As the examiner (C. H. B.) had not previously observed bodies of this type, he requested that another biopsy be made and unfixed tissue be submitted to the laboratory for cultural studies to determine whether or not they represented a form of fungus. While we were awaiting receipt of this material, fortu-

nately, Dr. Roger D. Baker, of the department of pathology, Duke University Medical School, Durham, N. C., a co-author [5] of a case report on chromoblastomycosis, was a visitor in the laboratory. On examination of the slide he readily expressed the opinion that the case was an instance of chromoblastomycosis.

On February 2 a second biopsy was performed, and material without fixative was sent to the laboratory by ordinary mail. It was ground up; part was treated with 95 per cent alcohol, and part was left untreated. Several Sabouraud plates were used. Some were incubated at room temperature and some at 37 C. No growth was obtained from the alcohol-treated specimen, but on the other plate within about ten days there appeared discrete colonies which exhibited a dirty gray color. Growth at room temperature was about the same as at 37 C. As the colonies became older, they darkened, and finally they were coal black. The general outline was circular; some reached a diameter of 1 to 2 cm., the older colonies being covered with gray furry material.

The diagnosis of chromoblastomycosis was therefore made. A culture was sent to the National Institute of Health for verification and for further study.

Our experience in failing to obtain a growth in the material which was treated with 95 per cent ethyl alcohol is similar to that of Weidman and Rosenthal,[2] who noted that growth was not obtained when cultures were planted with alcohol-treated inoculum.

The patient was released from the penitentiary on February 8. No further treatment could be followed by the hospital physicians. Electrocoagulation was recommended, but when the patient was last seen by a probation officer this recommendation had not been followed.

5. Martin, D. S.; Baker, R. D., and Conant, N. F.: A Case of Verrucous Dermatitis Caused by Homodendrum Pedrosoi (Chromoblastomycosis) in North Carolina, Am. J. Trop. Med. **16**:593 (Sept.) 1936.

MYCOLOGY

Chromoblastomycosis, as recognized at the present time, may be caused by any one of three fungi. Clinical variations in the disease are wide [5a] but are not correlated with differences in causation. Two fungi predominate in the causation of the disease, Phialophora verrucosa Medlar 1915 and Hormodendrum pedrosoi Brumpt 1922. The relationship of both fungi to one disease and gross resemblances between the two fungi have long suggested that they are genetically related,[6] but because of distinctive morphologic differences they have not been brought together into one genus. P. verrucosa is characterized by the semiendogenous formation of conidia in a cuplike structure, while H. pedrosoi produces larger spores acrogenously or pleurogenously and these conidia may occur in short branching chains. The nomenclature of the second fungus is somewhat confused, but the earliest bestowed name, Hormodendrum pedrosoi Brumpt 1922, has been generally used in the United States. There are, however, valid

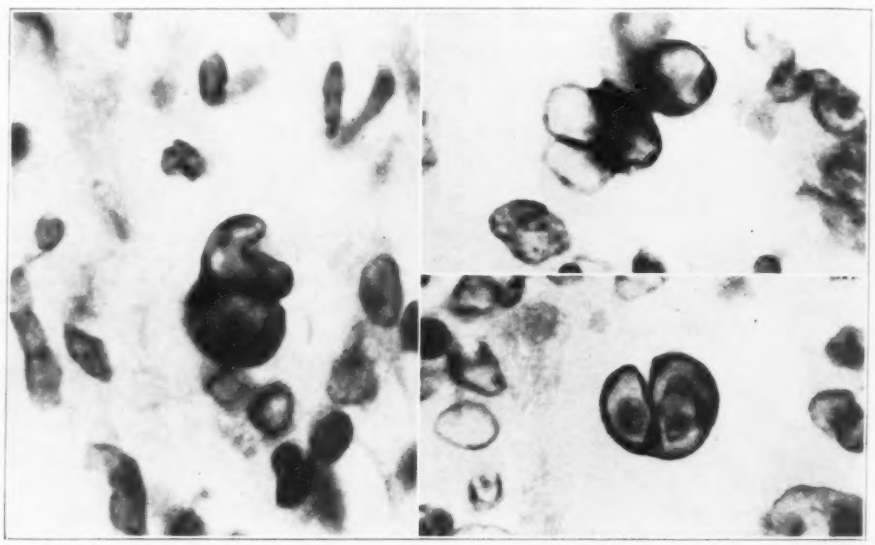


Fig. 3.—The cells of Phialophora pedrosoi within giant cells. Note the septate arrangement. Van Gieson's and Weigert's iron-hematoxylin stain; × 1500.

5a. Emmons, Hailey and Hailey,[1] Weidman and Rosenthal,[2] Pardo-Castello, Rio Leon and Trespalacios.[3]

6. (*a*) Wilson, S. J.; Hulsey, S., and Weidman, F. D.: Chromoblastomycosis in Texas, Arch. Dermat. & Syph. **27**:107 (Jan.) 1933. (*b*) Emmons, C. W., and Carrión, A. L.: The Phialophora Type of Sporulation in Hormodendrum Pedrosoi and Hormodendrum Compactum, Puerto Rico J. Pub. Health & Trop. Med. **11**:703 (June) 1936. (*c*) Weidman and Rosenthal.[2] (*d*) Pardo-Castello.[4] (*e*) Footnote 1.

taxonomic objections to the use of this name, Hormodendrum being actually a synonym of Cladosporium; and there appears to be some justification from a morphologic point of view for removing the fungus from that genus, branching chains of spores being short or lacking in most strains. Selection of the correct name is difficult because of the wide variation in methods of sporulation exhibited by the fungus.[7]

The first morphologic evidence of an actual genetic relationship between the two fungi was obtained in 1935 when the Phialophora type of sporulation was demonstrated sparsely but unmistakably in several strains of H. pedrosoi.[8] Complementary evidence of a close relationship was presented when Carrión[9] reported that the H. pedrosoi type of sporulation occurs rarely

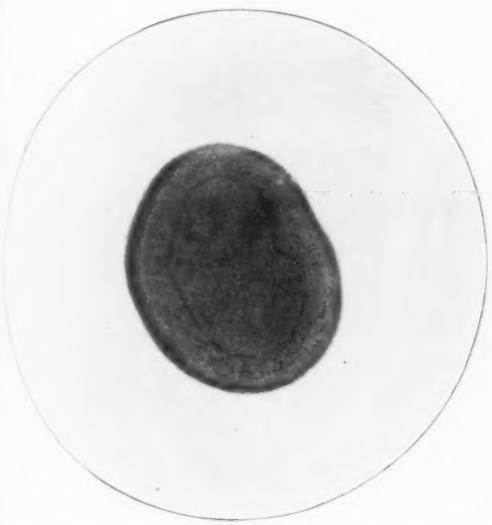


Fig. 4.—Phialophora pedrosoi. Culture on acid dextrose agar (Sabouraud) incubated four weeks at room temperature.

in one strain of P. verrucosa.[10] There is therefore a sound basis for recognizing a close relationship between the two fungi.

It is apparent from several recent publications that a change in the name of H. pedrosoi is necessary. Negroni[11] proposed the new name Fonsecaea pedrosoi. If no other name has priority there is considerable merit in this proposal which substitutes a new generic name for several older and unsuitable names and honors one of the South American investigators who have made notable contributions to the knowledge of this disease. Carrión, in a comprehensive and well illustrated report,[12] accepted this name, amending it, however, to include the strain of P. verrucosa previously mentioned. The strain in question is typical of P. verrucosa, producing spores which are possibly slightly larger than those of most strains of the species but are within the limits given by the original describer of the species. The difference in size of spores does not merit more than varietal rank, as Carrión pointed out in an earlier paper.[9] The occurrence of acropleurogenous sporulation is so rare that it can be demonstrated only with great difficulty and in a small proportion of preparations. One can hardly escape the expectation that an adequate search on suitable mediums would reveal it in other strains of P. verrucosa.

Carrión, therefore, so far as this one strain was concerned, actually brought together into one genus the two species H. pedrosoi and P. verrucosa. In choosing the generic name for this aggregate, he rejected the name Phialophora and selected the new name Fonsecaea. This appears to us to have been an error. It was equally necessary and equally easy to amend whichever name was to be used, but the rules of nomenclature require that the older of the two names be selected. It does not occasion any more confusion to broaden the definition of Phialophora than to broaden that of Fonsecaea. Thom,[13] in recommending this action, pointed out that a "generic name once established loses its etymologic limitations and becomes the designation of an aggregate rather than a unit or single species."

We have come reluctantly to the conclusion that the correct procedure is to recognize the close relationship between the two most important etiologic agents of the disease, a relationship which has been repeatedly suggested by investigators, by transferring H. pedrosoi to the genus

7. Emmons, C. W., and Carrión, A. L.: Hormodendrum Pedrosoi: An Etiological Agent in Chromoblastomycosis, Puerto Rico J. Pub. Health & Trop. Med. **11**:639 (June) 1936.

8. Carrión, A. L., and Emmons, C. W.: A Spore Form Common to Three Etiological Agents of Chromoblastomycosis, Puerto Rico J. Pub. Health & Trop. Med. **11**:114 (Sept.) 1935. Emmons and Carrión.[6b]

9. Carrión, A. L.: The Specific Fungi of Chromoblastomycosis, Puerto Rico J. Pub. Health & Trop. Med. **15**:340 (June) 1940.

10. This observation had been made independently by one of us (C. W. E.) in July 1939.

11. Negroni, P.: Estudio micológico del primer caso argentino de cromomicosis Fonsecaea (n. g.) pedrosoi (Brumpt, 1921), Rev. d. Inst. bact. **7**:419 (March) 1936.

12. Carrión, A. L.: Chromoblastomycosis, Mycologia **34**:424 (July-Aug.) 1942.

13. Thom, C.: Naming Molds, J. Washington Acad. Sc. **30**:49 (Feb. 15) 1940.

Phialophora. We accordingly propose the following emended definition of Phialophora and the necessary new combinations.

Phialophora Medlar 1915, *emend.*

Dematiaceous fungi characterized by either of two types of sporulation, viz., (1) semiendogenous production of colorless to faintly pigmented one cell spores within the cuplike mouth of a flask-shaped or tubular phialide (resembling spermatium formation of some Ascomycetes), or (2) acrogenous, pleurogenous or acropleurogenous production of brown one-celled conidia borne laterally on tuberculate processes of vegetative hyphae or, more commonly, on lateral or terminal coni-

Fonsecaea pedrosoi var. *phialophorica* Carrión Mycologia **34**:424, 1942

Phialophora pedrosoi Brumpt 1922 n. comb.

Syn: *Hormodendrum pedrosoi* Brumpt, Précis de parasitologie 1922

Fonsecaea pedrosoi Carrión Mycologia **34**:424-441, 1942

Phialophora compactum Carrión 1935 n. comb.

Syn: *Hormodendrum compactum* Carrión, Puerto Rico J. Pub. Health & Trop. Med. **10**:543-545, 1935

Fonsecaea compactum Carrión, Mycologia **34**:424-441, 1942

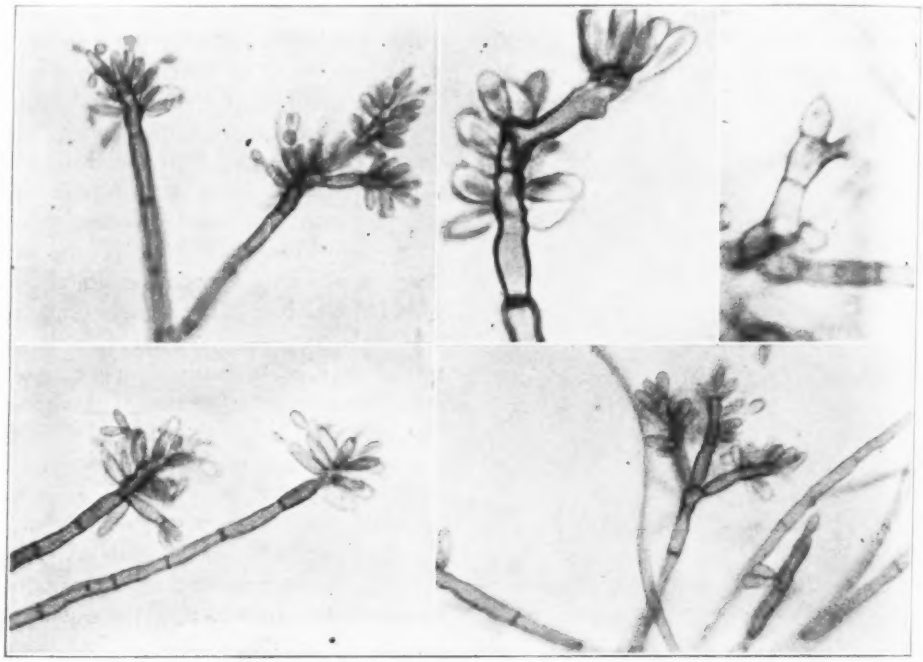


Fig. 5.—Phialophora pedrosoi. Methods of sporulation.

diophores and frequently catenate in short chains. Known species sporulate predominately or almost exclusively by one or the other method, but some strains of some species, in addition, sporulate rarely or aberrantly in the other manner.

Some species are etiologically associated with chromoblastomycosis in man. Others are known only from wood.

The known pathogenic species are:

Phialophora verrucosa Medlar, J. M. Research **32**:507-522, 1915

Syn: *Cadophora americana* Melin and Nannfeldt, Svenska skogsvårds tidskr. **32**:397-585, 1934 (See Conant [14])

Phialophora macrospora Moore and Almeida, Ann. Missouri Botan. Garden **23**:543-552, 1936

14. Conant, N. F.: The Occurrence of a Human Pathogenic Fungus as a Saprophyte in Nature, Mycologia **29**:597 (Sept.-Oct.) 1937.

The fungus isolated in the present case is typical of the species P. pedrosoi. The colony (fig. 4) is dark olivaceous, growing slowly on acid dextrose agar in the form of low dome, showing concentric zones of growth, the darker underlying mycelium being covered with short brownish to mouse gray hyphae. Microscopically the fungus produces brown elliptic spores borne laterally or terminally on short, denticulate, often crooked, septate, brown conidiophores (fig. 5). A few conidiophores of the P. verrucosa type were found. It is believed that this strain corresponds to the variety *communis* in Carrión's analysis of the species.[12] A further description would be merely a repetition of the many detailed descriptions of the fungus already published.

INTENSIVE AMBULATORY THERAPY OF SYPHILIS

THIRTY DAY MAPHARSEN TECHNIC

MAJOR SAMUEL GOLDBLATT

MEDICAL CORPS, ARMY OF THE UNITED STATES

Since the introduction of arsphenamine in the treatment of syphilis, many technics of abortive or intensive therapy have been proposed. Most of these methods have been rejected either because of inherent toxicity or because a "test of time" evaluation has demonstrated therapeutic failure.

This "test of time" evaluation of the results of therapy for syphilis is complicated by the characteristic long latent period and by the tendency of the disease to heal spontaneously in an unknown percentage of cases. Increased longevity resulting from the control of formerly fatal intercurrent diseases permits the later development of clinical and laboratory manifestations of syphilis in a group of patients who were formerly thought cured.

In the attempt to establish for syphilis a therapy of short duration which would rapidly render the patient noninfectious and be relatively nontoxic and potentially curative, Chargin and his associates [1] in 1933 devised an intensive treatment based on the speed shock theory of intravenous injections. They were finally enabled to administer as high as 1,200 mg. of mapharsen within five days by use of the slow intravenous drip. The treatment has been tried on a fairly large number of patients with infectious syphilis in several well conducted hospital clinics, with good results.

The increased frequency and severity of the toxic manifestations occurring with the five day drip method as compared with those with the usual conservative management led Rein and Wise,[2] Schoch and Alexander [3] and many other investigators to devise modifications which might prove to be more effective therapeutically and less toxic.

Early in 1941 I [4] treated a group of 100 previously untreated and unselected ambulatory patients with all types of syphilis by single intravenous injections of 100 mg. of mapharsen daily for twelve successive days. In order to reduce the number and severity of the reactive manifestations produced by this technic, I further modified the procedure to consist of the daily intravenous administration of 60 mg. of mapharsen dissolved in 5 cc. of sterile distilled water for thirty successive days. A total of 1,800 mg. of mapharsen was thus administered without adjuvant therapy. The first group of patients so treated was reported on in June 1941.[4]

EXPERIMENTAL DATA

The following report is concerned with clinical observations and laboratory investigations on another unselected group of 107 patients with syphilis of all types, treated by the intensive consecutive thirty day mapharsen method. The group consisted of 104 men and 3 women. The male population included 67 white men, 31 Negroes, 3 Mexicans, 2 Chinese and 1 Indian; the female group consisted of 1 Japanese woman and 2 white women. The ages varied from 16 to 55 years. With the exception of 1 white woman who was slightly underweight, the nutrition and development of all patients were good.

Diagnostic investigation included the history of previous diseases, data as to recent vaccinations and inoculations and physical examination with special emphasis on dermatologic, cardiovascular and neurologic phenomena. Urinalyses, dark field examinations, blood counts and determinations of blood chemistry, examination of the spinal fluid and roentgenologic, electrocardiographic and serologic examinations were obtained before treatment was instituted.

Syphilis in these patients was distributed as follows: Twenty-seven had primary lesions; 21 had secondary lesions and 17 had late active

1. Chargin, L.; Leifer, W., and Hyman, H. T.: Studies of Velocity and Response to Intravenous Injections: Application of Intravenous Drip Method to Chemotherapy as Illustrated by Massive Doses of Arsphenamine in Treatment of Early Syphilis, J. A. M. A. **104**:878 (March 16) 1935.

2. Rein, C. R., and Wise, F.: Mapharsen in Treatment of Syphilis in Office Practice, J. A. M. A. **113**: 1946-1952 (Nov. 25) 1939.

3. Schoch, A. G., and Alexander, L. J.: Intensive Arsenotherapy of Early Syphilis: Follow-Up Report of Ten Day Syringe Method of Treatment, Arch. Dermat. & Syph. **46**:128-129 (July) 1942.

4. Goldblatt, S., in discussion on papers of Drs. Leifer, Chargin and Hyman and Elliott and others, J. A. M. A. **117**:1165 (Oct. 4) 1941.

lesions; 7 had congenital syphilis, and in 35 the disease was latent.

Primary lesions had been present from three days to six weeks at the time of diagnosis. A single penile ulcer was observed in 15 patients, and multiple primary syphilitic ulcerations of the penis were noted in 8 patients. A single syphilitic ulcer accompanied by chancroidal and/or lymphogranulomatous lesions was found in 4 patients. Two intrameatal primary lesions and 2 chancres of the lip were observed.

Patients with secondary syphilis presented the following lesions: generalized maculopapular eruption, 6; mucous patches, 7; ulcerative syphilid, 3; extensive pustular eruption, 2; annular syphilid, 1; alopecia syphilitica with condylomata lata, 1, and iritis, 1.

The group with latent syphilis consisted of 32 men and 3 women. The duration of syphilis in this group varied from six months to twenty-five years. One young white man had undergone the five day drip treatment for primary syphilis eight months previously and had attained seronegativity after that therapy. At the time of my observation he was apparently suffering from a serorelapse. Four other men had received adequate treatment previously and were serologically fast. These patients were treated chiefly in an effort to determine the influence of this type of therapy on serologic fastness.

Lesions in the group with active syphilis consisted of: tabes dorsalis, 3; trifacial palsy of central origin, 1; moderately severe meningovascular involvement, 5; asymptomatic neurosyphilis, 4; early aortic aneurysm, 1; gumma, 2, and extensive nodular serpiginous syphilid involving the left shoulder, arm and thigh, 1. This last patient had concomitant moderately severe diabetes mellitus.

In the group with congenital syphilis 2 patients were completely asymptomatic, 2 had severe meningovascular involvement, 1 had bilateral tibial periostitis, 1 had separation of the tibial epiphysis and 1 had Hutchinson's teeth and nocturnal enuresis.

Twenty-seven of the patients with primary syphilis and 15 of those with secondary syphilis were subjected to dark field investigation. Treponema pallidum was demonstrated in penile lesions from 23 patients with primary syphilis and in cutaneous lesions from 12 with secondary syphilis. In the remaining 4 with primary and 3 with secondary syphilis dark field examination gave persistently negative results in spite of numerous examinations.

The blood serum was examined by Kahn and Kolmer-Wassermann methods simultaneously in two separate laboratories. All positive reports were confirmed by retest of another sample of serum drawn one day after receipt of the positive report. If negative reports were obtained, the serum was retested weekly during the period the patient was under treatment and every two weeks of the entire period of observation. Estimations of the Kahn titer were obtained whenever possible.

Twelve patients with primary syphilis had negative serologic reactions, and 15 had positive reactions; the reactions of all patients with secondary syphilis were positive. Serologic reactions were positive in 14 and negative in 3 of the group with late active syphilis. Of the patients with congenital syphilis 1 had doubtful and 6 had positive serologic reactions.

The spinal fluid was examined in all cases immediately after demonstration of spirochetes by dark field examination or after confirmation of a positive serologic reaction. One showed a doubtful positive serologic reaction and 8 had definite abnormalities in the serologic reaction, colloidal gold curve, protein content and cell count, while results for 98 were completely negative.

Investigation of the formed elements of the blood revealed only slight abnormalities. A tendency to leukocytosis in patients with early syphilis and a shift toward relative lymphocytosis in patients with late syphilis were noted.

The blood chemistry was investigated routinely with reference to urea nitrogen, sugar, chlorides, cholesterol, carbon dioxide–combining power and icteric index. For 30 per cent of the patients selected at random the levels of nonprotein nitrogen, calcium, serum albumin and serum globulin were determined. Carbon dioxide–combining power and sugar content were within the average normal range, regardless of the duration or activity of the syphilis. Chlorides were depressed in the direction of low normal values, while the nonprotein nitrogenous elements and icteric index tended toward high normal limits. The cholesterol content was found to exceed the high normal values in some patients with involvement of the central nervous system, while in patients with other types of syphilis the average normal range was observed. The calcium content and the protein constituents of the blood as well as the albumin-globulin ratio were found within average normal levels.

Glycosuria was noted once and albuminuria six times. Formed elements were found in the urine only of patients with active gonorrheal urethritis.

Roentgenograms of the chest with special reference to the cardiovascular system were obtained in 99 cases. One patient had hypertensive heart disease; 1 had mitral disease; 1 had a mild aortic

aneurysm, and 1, a noncharacteristic right-sided cardiac hypertrophy.

Roentgenograms of the bones and joints revealed separation of the tibial epiphysis in 1 case and bilateral tibial periostitis in another.

Electrocardiograms were obtained in 70 random cases. Mild changes suggestive of slight myocardial damage were noted in 2; readings indicating auricular hypertrophy were reported once, and 1 cardiogram was interpreted as abnormal but noncharacteristic. Sixty-six cardiograms were completely normal.

Routine use of chick embryo (lygranum) and human antigens for Frei tests yielded 30 per cent positive reactions. A positive Mantoux reaction was obtained with purified protein derivative in 35 per cent of the patients.

Gonorrheal urethritis was noted in 22 per cent of the entire group. It was evenly distributed throughout, in contrast with chancroid, which was encountered in only 3 per cent, all of which, however, occurred in association with primary syphilis.

TREATMENT

An intravenous injection of 60 mg. of mapharsen dissolved in 5 cc. of sterile distilled water was given each morning, including Sundays, for thirty consecutive days. Before each treatment the patient was stripped, carefully examined and closely questioned about reactions from his previous treatments.

With this therapy the patients noted an increase in appetite. A gain in weight of from 5 to 15 pounds (2.3 to 6.8 Kg.) was observed in over 80 per cent of the patients treated. A feeling of well-being was almost universally described after the tenth to the fifteenth injection. Loss of weight was not observed; a few patients failed to increase their weight in spite of an alleged increase in appetite.

A small number of patients suffering from acute and from chronic alcoholism were treated in this series. These patients reacted favorably to the intensive treatment.

Forty patients received a sulfonamide compound in 6 Gm. daily doses concurrently with the daily injections of mapharsen for the treatment of an associated gonorrhea, chancroid or lymphogranuloma venereum. Both drugs were well tolerated, no adverse reactions due to either medication being noted during or after treatment.

Complete healing of all lesions in patients whose disease was in the early infectious stage occurred in an average of ten days.

Albuminuria previously noted in 6 patients disappeared during treatment. One patient with congenital syphilis showed a mild transient albuminuria immediately after treatment. Glycosuria noted in the diabetic patient disappeared after treatment with insulin.

The serologic reactions of patients who had previously attained seronegativity remained negative. With 4 exceptions all patients with positive reactions showed a definite reduction in the serologic titer. The 4 patients with latent syphilis and serologic fastness showed a reduction to complete negativity.

A study of spinal fluids of 94 patients immediately after therapy revealed that all fluids which were normal preceding treatment remained normal. Half of the abnormal fluids became normal at the conclusion of treatment.

A consistent depression of the erythrocytes amounting to from 2 to 4 per cent was noted after treatment. In instances in which leukocytosis was initially found, the white cell count was depressed to within normal limits; normal white cell counts were not significantly changed. A tendency to slight depression of granulocytes and increase in percentage of lymphocytes without change of any sort in number of eosinophils was noted.

The nitrogenous elements of the blood, carbon dioxide–combining power and icteric index remained at pretreatment levels, while the sugar and chloride contents were decreased slightly. The influence of intensive therapy on the calcium and the protein content of the blood was studied in 32 unselected patients. With the exception of slightly increased values for calcium, no significant changes occurred after treatment.

Repeated electrocardiographic studies on 62 patients immediately after termination of treatment revealed that the records for the 58 who had previously had normal records had remained normal; those for the patient with previous myocardial damage and the 1 with aurciular hypertrophy were unchanged. The patient with noncharacteristic changes on previous examination now showed sinus bradycardia. One Negro man with syphilis of the central nervous system whose electrocardiogram before treatment was normal revealed a slightly prolonged auriculoventricular conduction time.

Roentgenograms of the chest for 84 patients on termination of treatment showed no progressive or toxic changes even in patients with previous evidence of cardiovascular damage. Roentgen studies of the bones and joints revealed definite improvement in lesions of these structures at completion of therapy.

TOXIC REACTIONS

There were no fatalities or severe reactions of any type. There was no clinical evidence of hemorrhagic encephalitis, peripheral neuritis or

renal or hepatic damage. No instances of exfoliative dermatitis, blood dyscrasias, shock or nitritoid crises or activation of other latent diseases were noted.

In 2 white men with early secondary syphilis Jarisch-Herxheimer reactions with accentuation of secondary lesions and moderately severe fever developed four hours after the first injection. Treatment was continued for both patients on the day following the reaction and was completed without incident.

One young white man with secondary syphilis had a toxic macular erythema after the ninth injection. This was accompanied by suffusion of the mucous membranes and a temperature of 104 F. This syndrome subsided in three days, but the patient reacted with a slight increase in temperature and malaise lasting twelve hours after each succeeding injection of mapharsen, even in greatly reduced dosages.

In 1 white woman aged 25 with latent syphilis of unknown duration a toxic erythema which cleared spontaneously in twelve hours developed after the ninth injection. The succeeding injection produced abdominal cramps, a more widespread macular erythema and a temperature of 100 F. This syndrome cleared in twenty-four hours, but it recurred after the next injection, even with a reduced dose. The serologic reactions of both patients were reversed immediately after these episodes. The inability of these patients to continue arsenical therapy with impunity suggests that both were suffering from a mild arsenical reaction rather than the classic erythema of Milian.

Mild nausea, occasional vomiting and mild diarrhea were encountered. These gastrointestinal manifestations were comparatively rare, and they disappeared in the susceptible patients as treatment progressed. It was not necessary at any time to interrupt or terminate treatment because of gastrointestinal reactions.

RELAPSE AND PROGRESSION

During the entire period of observation no patient manifested mucocutaneous relapse or neurorecurrence. One Negro man originally treated by the thirty day method was returned to the hospital three months later with multiple ulcerations of the penis which proved to be extensive primary lymphogranuloma venereum. During the investigation of his case it was noted that the Kahn titer, which was previously static, began to rise. This patient was subjected to a second course of thirty days' treatment, which was tolerated without incident.

Two patients with severe syphilis of the central nervous system sustained the first course of treatment without reaction. The decrease in serologic titer was decided but incomplete after three months of observation. At the end of this time the titer began to rise and the patients were subjected to a second course of treatment. The daily dose of mapharsen (60 mg.) which each received was accompanied on alternate days by the intraveous injection of 1 cc. of typhoid vaccine in 1,000 cc. of isotonic solution of sodium chloride, the dose of mapharsen being administered at the height of the fever. Each patient sustained fifteen separate attacks of fever, the temperature ranging from 102 to 105 F., while the retreatment course was given. This treatment was well tolerated. The tonic effect of mapharsen when so used stimulated the appetite to such an extent that the depressive effects of the prolonged fever were to a large extent immediately counteracted.

Tolerance for this repeated course seemed to be equally as good as for the original course. The therapeutic value of retreatment courses, however, cannot be estimated at present.

SUMMARY

A group of 104 men and 3 women were subjected to intensive arsenical therapy consisting of injection of a total dose of 1,800 mg. of mapharsen. This was administered intravenously in daily doses of 60 mg. for thirty consecutive days.

The serologic reactions, which were positive in 81 per cent of the total group before treatment, showed a decided and continuing reduction in titer. On termination of treatment positive serologic reactions were noted in 55 per cent, one month later in 40 per cent and two months later in 35 per cent of the patients. Follow-up investigations from three to six months after termination of treatment indicated that the reactions of 20 per cent remained positive. The reactions of an additional 8 per cent were reported as doubtful. All patients who began treatment with a negative serologic reaction retained their seronegative status during this entire period of observation. During this six month period, with but 1 exception the patients with infectious and with latent syphilis whom I was able to follow had complete serologic reversal. Three patients, 1 of whom was the exception previously noted, suffered serologic relapses. None of these had attained seronegativity, but all showed an increase in titer after a three month period of static or decreasing titer following treatment.

The spinal fluids of 9 patients gave positive evidence of syphilis originally. Six became negative and 3 remained persistently positive for the disease six months after termination of treatment.

No significant changes were observed in the formed or chemical constituents of the blood.

Electrocardiographic examination revealed no deleterious effects on cardiac function, either immediately or remotely.

Roentgenographic examination of the cardiovascular system indicated no progressive or toxic sequelae.

Retreatment was carried out for the 3 patients with serologic relapse. Two with severe meningovascular involvement sustained fifteen attacks of induced fever, concurrent with the hyperintensive therapy, without untoward reaction.

It was found possible to administer sulfonamide compounds in normal doses for the treatment of intercurrent disease without interrupting the intensive mapharsen therapy.

Alcoholism apparently did not constitute a contraindication to intensive treatment.

Complete healing of all the lesions of infectious syphilis required an average of ten days. A gain in weight of from 5 to 15 pounds (2.3 to 6.8 Kg.) and increased appetite and feeling of well-being were noted in the large majority of patients. No mucocutaneous relapse or neurorecurrence developed during the period of observation in this series.

Reactions to treatment consisted of two of the Jarisch-Herxheimer type and two of mild arsenical reaction in some aspects simulating the Milian ninth day erythema type.

Gastrointestinal upsets were rare and mild and disappeared as treatment progressed.

Various factors have intervened to prevent long-continued observation after the completion of treatment. The attempted use of the questionnaire method as a substitute for personal observation proved unsatisfactory.

The comparatively small number of patients treated and the short period of observation limit the conclusions here presented to a consideration of the immediate and the remote toxicity of the method. The clinical value of this therapeutic approach for the various types of syphilis treated cannot be finally estimated for some years.

CONCLUSIONS

A method of intensive and possibly abortive therapy for syphilis (administration of mapharsen daily for thirty days) which seems to be applicable to the treatment of the disease in its common phases was devised. This therapy appears to be nontoxic, either remotely or immediately. Hospitalization is not usually required, and the treatment is inexpensive, is easily available and does not disrupt the economic life of the patient. Naturally, if this technic is adopted, the patient should be followed closely and carefully examined at each visit in order to detect reactions at the earliest possible moment. This technic should be employed only by an expert.

ULCERATIVE DIPHTHERIA OF THE SKIN

SPORADIC AND EPIDEMIC TYPES OBSERVED IN HAIFA, PALESTINE

S. GILL, M.D.
HAIFA, PALESTINE

Chronic ulcerative diphtheria of the skin occurs sporadically in Palestine in persons weakened by infectious disease or with lowered nutrition. In the dermatologic department of the Hadassah Policlinic in Haifa, there was 1 case of the disease in 2,000 cases of dermatologic diseases. The lesions were almost invariably located on the legs.

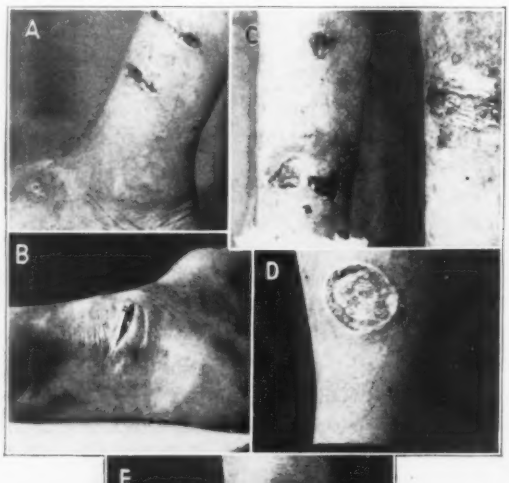


Fig. 1.—*A*, ulcerating diphtheria of the skin of three years' duration in a boy aged 13 years. *B*, multiple diphtheritic ulcers (total of 13) of eighteen months' duration in a boy aged 15 years. *C*, diphtheritic ulcer of five days' duration in a boy aged 7 years. The lesion resembles a ripe open seed shell. *D*, acute diphtheritic ulcer of eighteen days' duration in a girl aged 17 years. *E*, appearance of the scar above the ankle one year after healing of a diphtheritic ulcer in a girl aged 15 years.

Epidemic ulcers affected members of the British Army in Palestine during World War I.[1] Craig in 1916 found Corynebacterium diphtheriae in a great part of them. I do not know whether at the same time civilians also suffered from acute diphtheritic ulcers.

In 1940 an outbreak of cutaneous diphtheria occurred in the army in northern Palestine. Two years later, in August 1942, a small epidemic of diphtheritic ulcers occurred among the civilian population.[2] During a period of four months 22 cases were observed, and eight months later there were 17 new cases.

GENERAL DESCRIPTION

In 12 cases there were multiple ulcers; in the others the lesions were single. In all except 2 cases, the ulcers were situated on the legs. The

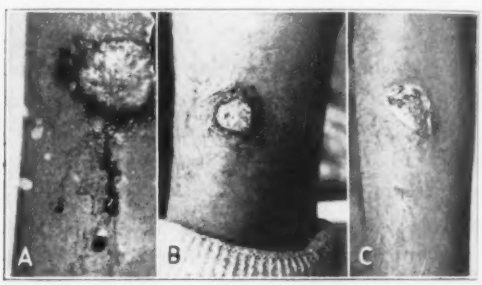


Fig. 2.—*A*, pustule of a nondiphtheritic infection (desert sore), showing spontaneous opening at periphery with abundant sanguineous discharge. *B*, nondiphtheritic ulcer (desert sore) after removal of the pustule with a tampon. *C*, nondiphtheritic ulcer (desert sore) of three weeks' duration in a boy of 17 years.

size of the ulcers varied considerably, the largest being 8 by 4 cm. In the 39 cases of the two series observed, C. diphtheriae was cultured on Löffler's medium.

Unfortunately, only a few patients applied for treatment during the first days of the infection. They reported that at the outset the affected area was bitten by an insect or was the site of a slight trauma. In 3 cases, I saw an open pustule appearing as if it had been sharply cut in the middle, resembling a seed pod which was ripe and open. A serous or sanguinolent discharge from the pustule was minimal at this stage. In

1. Veld Sore, in Manson, P.: Manson's Tropical Diseases, edited by P. H. Manson-Bahr, Baltimore, William Wood & Company, 1940.

2. Cameron, J. D. S., and Muir, E. G.: Cutaneous Diphtheria in Northern Palestine, Lancet **2**:720 (Dec. 19) 1942.

1 case the swelling surrounding the pustule was 8 cm. in diameter. During the first days there was a painful adenitis, mostly of the femoral nodes, none of which suppurated

Five patients applied for treatment at the very beginning of the disease. At that time the necrotic epidermis and debris of the pustule hung on the edge of the ulcer, covering it with thin small pieces, at times of a clear white or blue color. It is not exact to call the lesion an ulcer at this stage, as there was only a necrotic transformation of the skin into a pseudomembrane. In 2 cases this was as white as gypsum; in others the color ranged from gray to a bluish red, depending on the amount of blood. The pseudomembrane, adherent to the subcutis, later separated from it piece by piece. After the separation of the pseudomembrane, there remained a more or less typical punched-out ulcer with, at times, rolled edges. Ulcers older than six weeks caused little or no pain. After the surrounding inflammation subsided, the edges of the ulcers tended to become callous.

DESERT SORE

During the same period when the diphtheritic ulcers occurred, another type of ulcer was seen, often in the same family. These ulcers were clinically similar to those of cutaneous diphtheria, but C. diphtheriae was never isolated from them. The organisms routinely found in these lesions were staphylococci, streptococci and occasionally Bacillus xerosis. There were about 100 cases of these ulcers.

It is questionable whether there is any etiologic relationship between desert sore and the acute diphtheritic ulcer. Of 1,000 cases of desert sore encountered in the western desert, only 2 proved to be due to infection with Löffler organisms.[3]

3. Rapport, H. M.: Desert Sores, Brit. M. J. **2**:96 (July 25) 1942.

TREATMENT

An injection of antidiphtheritic serum (10,000 units) was given in only 1 case. In 23 of the 39 cases observed, the serum was applied locally. The fluid was dropped on the ulcer from a syringe. The ulcer was then closed with a piece of gauze soaked in the serum, covered with parchment paper and fixed with adhesive plaster. Over this, a large bandage was placed for twenty-four hours.

In 16 cases, I found it sufficient to dispense with the serum and apply first a lotion, and then an ointment containing sulfapyridine, 10 per cent methylthionine chloride and 2 per cent gentian violet (methylrosaniline). The same remedies were used to continue treatment in cases in which the serum had been applied locally, usually during the first six or eight days. The skin around the ulcer was covered with zinc oxide paste.

Daily observation of the ulcers was necessary, as not all behaved in the same manner. A quick change of treatment brought good results, the methods including the use of antidiphtheritic serum, alibour water (water containing zinc and copper sulfate), measures to prevent periulcerative dermatitis, cautious application of ointments for epithelization, and ultraviolet irradiation.

The duration of the treatment varied from two weeks to three months, depending on the age and size of the ulcers. There were 2 recurrences, 1 occurring three weeks after complete epithelization and 1 two months after it.

No diphtheritic paralysis occurred in any of the 39 patients. This was due, in my opinion, first, to early diagnosis, and, second, to prolonged neutralization of the diphtheritic toxin by repeated local applications of serum.

36 Herzl Street.

HEREDITARY ECTODERMAL DYSPLASIA

REPORT OF A CASE, WITH EXPERIMENTAL STUDY

ZACHARY FELSHER, M.D.

CHICAGO

Charles Darwin, in his work "The Variations of Animals and Plants Under Domestication," [1] mentioned a communication from Wedderburn, wherein he described 10 men of a Hindoo family with little hair on the body, early baldness, partial anodontia and excessive dryness of the skin in hot weather. He observed that this anomalous condition did not occur in the female members of the family but was transmitted to males by healthy female conductors. Wedderburn reported this case in 1838. In 1848 Thurman [2] described 2 similar cases, and in 1883 Guilford [3] reported another.

The total number of cases of hereditary ectodermal dysplasia known is difficult to estimate. Lord and Wolfe [4] (1938) collected about 40 cases from the literature. De Silva [5] (1939) mentioned 48 cases, excluding the Hindoo cases of Wedderburn (10) and the cases reported by Thadani [6] (2) in 1921. Clouston [7] (1939) differentiated two clinical types of hereditary ectodermal dysplasia; first, the more common, "hidrotic" type, with defects of teeth and hair but without hypoplasia of sweat glands and, second, the rarer, "anhidrotic" type, with absence of sweat glands in addition to other ectodermal defects. He reported on over 50 cases of the first type that he had encountered and only 4 of the second. Since de Silva's [5] review, about 13 more cases of the anhidrotic type have been described.[8]

In 1921 Siemens,[9] on the theory that combined defects of hair, teeth and sweat glands are a sex-linked recessive character transmitted by females who are never affected, called it "anidrosis hypotrichotica sexoligata." In a later communication (1937) Siemens [10] stated that partial ectodermal defects also occur in women, and he dropped the term "sexoligata." Weech [11] (1929) reported a case of such defects in a girl and coined the now widely used name, "hereditary ectodermal dysplasia," for this condition. Weech's patient, however, showed no absence of sweating. In general, of the few females affected still fewer are reported as having gross defects of the sweat glands. I could find in the literature only 4 such cases of patients actually seen by the writers. These were the 2 sisters reported on by Walter Schär [12] (1927) and 2 other women in a family described by

From the Section of Dermatology and Syphilology, Department of Medicine, University of Chicago.

1. Darwin, C.: The Variations of Plants and Animals Under Domestication, New York, D. Appleton & Sons, 1892, vol. 2, p. 319.

2. Thurman, J.: Two Cases in Which the Skin, Hair and Teeth Were Very Imperfectly Developed, Proc. Roy. Med. & Chir. Soc. **31**:71, 1848.

3. Guilford, S. H.: A Dental Anomaly, Dent. Cosmos **25**:113, 1883.

4. Lord, L. W., and Wolfe, W. D.: Hereditary Ectodermal Dysplasia of the Anhidrotic Type, Arch. Dermat. & Syph. **38**:893 (Dec.) 1938.

5. de Silva, P. C. C.: Hereditary Ectodermal Dysplasia of the Anhidrotic Type, Quart. J. Med. **32**:97, 1939.

6. Thadani, K. I.: A Toothless Type of Man, J. Hered. **12**:87, 1921.

7. Clouston, H. R.: The Major Forms of Hereditary Ectodermal Dysplasia, Canad. M. A. J. **40**:1, 1939.

8. (*a*) Glass, L. C., and Yost, D. H.: Inherited Inability to Sweat, J. Hered. **30**:477, 1939. (*b*) Cook, T. J.: Hereditary Ectodermal Dysplasia of the Anhidrotic Type, Am. J. Orthodontics **25**:1008, 1939. (*c*) Sunderman, F. W.: Persons Lacking Sweat Glands, Arch. Int. Med. **67**:846 (April) 1941. (*d*) Lowenburg, H., Jr., and Grimes, E. L.: Ectodermal Dysplasia of the Anhidrotic Type, Am. J. Dis. Child. **63**:357 (Feb.) 1942. (*e*) Lewin, M. L.: Nasal Deformity in Anhidrotic Ectodermal Dysplasia, Arch. Otolaryng. **35**:210 (Feb.) 1942. (*f*) Stadler, H., and Blackstone, C. H.: Hereditary Ectodermal Dysplasia of the Anhidrotic Type, J. Pediat. **21**:229, 1942. (*g*) Brodie, A. G., and Sarnat, B.: Ectodermal Dysplasia (Anhidrotic Type) with Complete Anodontia: Serial X-Ray and Cephalometric Appraisal, Am. J. Dis. Child. **64**:1046 (Dec.) 1942. (*h*) Bloom, D.: Hereditary Ectodermal Dysplasia of the Anhidrotic Type Associated with Syndactylia and Lichen Nitidus, Arch. Dermat. & Syph. **43**:900 (May) 1941.

9. Siemens, H. W.: Ueber recessive geschlechtsgebundene Vererbung bei Hautkrankheiten, Arch. f. Dermat. u. Syph. **136**:69, 1921.

10. Siemens, H. W.: Studien über Vererbung von Hautkrankheiten: XII. Anidrosis hypotrichotica, Arch. f. Dermat. u. Syph. **175**:565, 1937.

11. Weech, A. A.: Hereditary Ectodermal Dysplasia, Am. J. Dis. Child. **37**:766 (April) 1929.

12. Schär, W. V.: Das Kieferwachstum bei angeborenem Zahnmangel und vollständiger Zahnlosigkeit (Anodontia Congenita), Schweiz. Monatschr. f. Zahnh. **37**:559, 1927.

Glass and Yost[8] (1939). Roberts[13] (1929) mentioned 3 females who were said to have patches on their bodies that did not sweat. In any event, the predominance of males over females with defects of sweat glands has been striking in the cases hitherto reported.

The inheritance of hereditary ectodermal dysplasia is evidently not as clearcut as was first thought. Cockayne[14] (1933) expressed the belief that there are two types of inheritance, dominant and recessive. Clouston[7] claimed that the dominant, not sex-linked, type of inheritance occurs rather frequently and that the patients are characterized by defects of hair, teeth and nails, without aplasia of sweat glands. For the rarer type, with anhidrosis, inheritance was held as sex-linked recessive. Reports of changes in the nails have been few in cases of the latter type, and in addition to defects of the sweat glands there have often been aplasia of the mucous glands of the mouth and nose associated with ozena. Levit[15] (1936) opposed the view that there are two different types of inheritance. On the theory that the great majority of pathologic mutations in man are neither fully dominant nor fully recessive, he explained the inheritance of ectodermal dysplasia as due to conditionally dominant genes with sex linkage. Such genes would show their effect in at least some heterozygous females. Siemens[10] (1937) analyzed from the literature records for nineteen affected families (with complete registration of siblings). He concluded that the defects could not be due to the same gene but were attributable to several allele genes of different dominant strengths, one part of these being intermediately sex linked. He also found that keratosis follicularis spinulosa decalvans has a similar type of heredity, this disease and "anidrosis hypotrichotica" being the only two diseases of the skin with such unique inheritance.

REPORT OF CASE[16]

R. S., a 17 year old student born in Germany, had known since childhood of the absence of certain upper and lower teeth. A similar condition in his brother and in other members of the family was also known. Otherwise his past history had been noncontributory until two years previous to the examination. At that time the patient traveled from northern France to Spain. During a short residence there, before coming to the United States, he noted that when the outdoor temperature went over 85 F. he would begin to have a body temperature often rising to 104 F. Accompanying the elevation in temperature, the patient complained of weakness, fatigue and occasional vertigo. He obtained relief by cold applications and showers. On his arrival in the United States, one year previous to examination, he continued to have the same symptoms. It should be noted that before going to Spain he had always lived in the temperate climates of southern Germany and northern France, where the outdoor temperature rarely went above 85 F.

The physical examination revealed several characteristic conditions. The facies showed a remarkable resemblance to that of other patients with hereditary ectodermal dysplasia, for instance, to those of Janitzkaia and Riabov.[17] The parietes were prominent, the jaw small and pointed and the forehead high (fig. 1). The skin was fine and dry. The palms were conspicuously free of any moisture. Small pigmented spots were found on the forehead, nose and cheeks. Follicular hyperkeratoses were present on the chest. The nails were normal.

Fig. 1.—Facies of patient, showing scanty hair of the scalp and eyebrows and aplastic lower jaw.

There was no saddle nose or rhinitis, as found in many other patients with ectodermal dysplasia. Lacrimation and salivary secretion were normal, as were the senses of taste and smell. The hair of the scalp was fine in texture and diffusely scanty. The eyebrows and the hair of the axillas and pubes were similar. The eyelashes were absent, but the beard was normal. On the chest a few downy hairs could be seen. The nipples and the mammary tissues were normal.

The teeth showed a good deal of dental repair, the exact details of which were not known to the patient. As far as could be determined from the history and the roentgen examination, there was a congenital absence of the upper and lower lateral incisors, lower first incisors, upper and lower first and second bicuspids and upper and lower third molars. The upper first incisors showed a tendency toward peg shape but were crowned.[18]

13. Roberts, E.: The Inheritance of Anhidrosis Associated with Anadontia, J. A. M. A. **93**:277 (July 27) 1929.

14. Cockayne, E. A., cited by Siemens.[10]

15. Levit, S. G.: The Problem of Dominance in Man, J. Genetics **33**:411, 1936.

16. The patient was presented before the Chicago Dermatological Society on Oct. 20, 1943.

17. Janitzkaia, E., and Riabov, M.: Ein ausgebreiteter Ektodermaldefekt, Ztschr. f. klin. Med. **107**:381, 1928.

18. The dental examination was made by Dr. J. R. Blayney, Zoller Dental Clinic, University of Chicago.

The family tree, as obtained from data given by the patient, shows a sex-linked type of inheritance, with affected males and female conductors (fig. 2).

EXPERIMENTAL STUDIES

Although it is generally assumed that the anhidrotic type of ectodermal dysplasia is associated with complete absence of sweat glands, it has not been experimentally demonstrated that these glands are completely absent. Small or medium-sized biopsy specimens usually show no sweat glands, but obviously such absence may also occur in partial defects. Sunderman [8c] (1941) in a biopsy of an axilla noted the absence of eccrine glands, as contrasted to the presence and excellent development of the apocrine glands. Elevation of the body temperature at high external temperatures (hyperthermia) indicates that the number of functioning sweat glands is insufficient

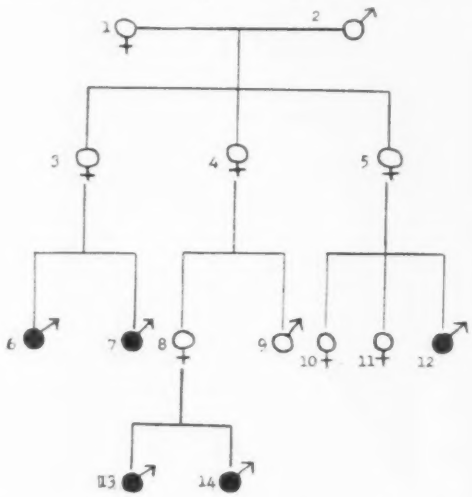


Fig. 2.—Family tree of R. S. Sex-linked inheritance with affected males and healthy female conductors. *1* and *2*, involvement unknown. *6*, dentition and hair defective; involvement of sweat glands unknown. *7*, dentition probably defective; hair said to be normal; involvement of sweat glands unknown. *12*, dentition defective; involvement of hair and of sweat glands unknown. *14*, hair and dentition defective; involvement of sweat glands unknown. *13*, the patient.

for physical regulation of heat, but this does not connote complete absence of sweat glands, either. I therefore attempted to visualize secretion of sweat directly in my patient in order to determine whether the defect was partial or complete.

Effect of Heat.—Minor's [19] method was used to visualize secretion of sweat. This method is based on the iodine-starch reaction, which takes place only in the presence of moisture. If the dry skin of the patient is painted with Minor's iodine solution [20] and after the solution has dried is covered with fine starch powder, sweat droplets will appear as bluish black puncta. Sweating was induced by putting the patient into a heat chamber placed over his bed. Body temperature was recorded orally. In a fifty-five minute experiment in which the temperature of the air within the chamber rose from 84 to 130 F., the body temperature rose from 98.4 to 101 F. A normal control subject under similar conditions showed no rise in body temperature.

Fifteen minutes after being put into the heat chamber, the patient began to show isolated spots of sweating in the axillas, on the upper part of the chest and about the nipples. Confluence of the sweat drops proceeded rapidly in the axillas but less rapidly about the nipples and on the chest, where sweat was also seen. It was evident that the usual sites of apocrine glands showed normal function. The secretion of the eccrine glands over the entire surface of the body was greatly diminished. The face and lower part of the abdomen showed practically no secretion. The remaining areas, excluding the sites of apocrine glands, showed only isolated spots of sweat (fig. 3).

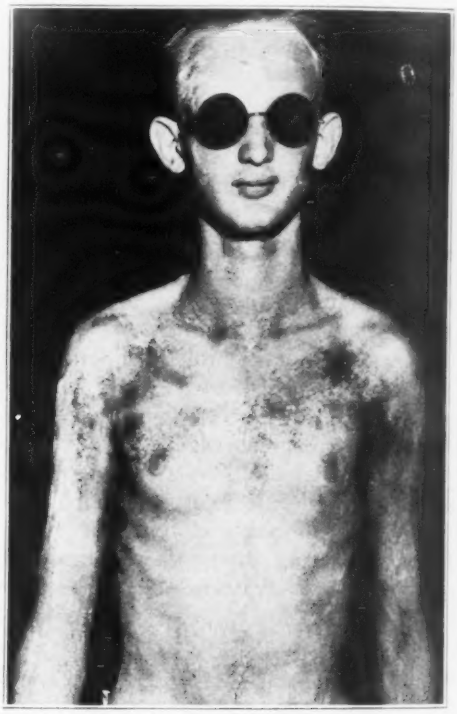


Fig. 3.—Close view showing confluence of sweat droplets in areas of apocrine glands (axillas, upper part of chest and perimammillary regions).

An area on the left forearm measuring about 72 square centimeters was marked off and sweating visualized by Minor's method. A close picture of this area showed 405 blue puncta corresponding to the pores of functioning sweat glands (fig. 4). Normal figures for this area are approximately 8,000, as calculated from the results of Krause [21] for healthy persons. The number of sweat glands is thus about 5 per cent of normal. On an area of the left thigh measuring about 122 square

19. Minor, V.: Ein neues Verfahren zu der klinischen Untersuchung der Schweissabsonderung, Deutsche Ztschr. f. Nervenh. **101**:302, 1927.

20. Iodine, 4 Gm.; castor oil, 20 cc., and absolute alcohol, 180 cc.

21. Krause, cited by Kuno, Y.: The Physiology of Human Perspiration, London, J. & A. Churchill, Ltd., 1934, p. 7.

centimeters 476 puncta were counted. The corresponding normal figure is approximately 6,800. The number of sweat glands here is 7 per cent of normal.

Effect of Acetylcholine.—A suitable method of testing the skin for the presence of functioning sweat glands is by means of intradermal injection of solutions of acetylcholine. This method was used by Kahn and Rothman [22] in their experiments on sympathectomized patients. Under the influence of nervous impulses, acetylcholine is liberated physiologically at the peripheral endings of the sweat nerve fibers and acts as a chemical mediator directly on the cells of the sweat glands.[23] If acetylcholine is injected intradermally, it stimulates the sweat gland cells in the area of its diffusion.

The skin of the patient's forearm and simultaneously that of a normal control person were prepared with Minor's iodine solution and starch powder. One-tenth

Fig. 4.—Left forearm, showing isolated spots of sweat secretion and great diminution in number of eccrine glands.

cubic centimeter of a 1:100 solution of acetylcholine bromide was injected intradermally into each subject. The normal subject showed a striking response over an area of 5 cm. in diameter, with confluence of the droplets in the center; the patient showed hardly any visible spot of secretion (fig. 5).

Measurement of the Insensible Perspiration.—The insensible perspiration was measured by absorption of water vapor with anhydrous calcium chloride over a circumscribed area of the skin. Small linen bags were filled with the calcium chloride, weighed and suspended on hooks under the domes of glass bells, with a diameter of their lower open surface of 4.71 cm. The bells were firmly fixed to the skin by strips of waterproof adhesive tape and allowed to remain attached for exactly one hour (fig. 6). The increase in weight of the calcium chloride bags indicated the amount of insensible perspiration. The bags were weighed before and after the experiment in weighing flasks with ground glass stop-

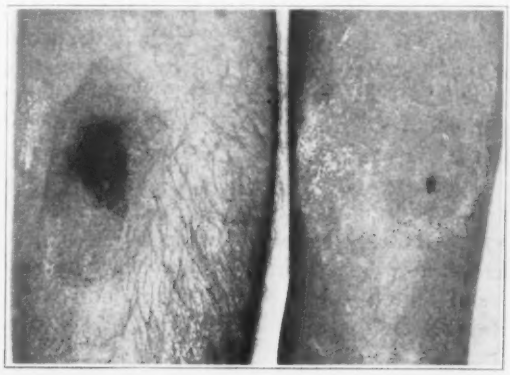


Fig. 5.—Response of a normal test person (left) and of the patient (right) to 1:100 acetylcholine bromide intradermally. The small confluent area on the patient's forearm is an artefact caused by reflux of the solution through the needle puncture.

pers and were transported to and from the balance room in a desiccator. Suspension on and removal from the glass hooks of the bells were performed with clean dry steel forceps. After some training this transfer was made within a few seconds, and any absorption of moisture from the air during the manipulation became negligible. Under these conditions parallel values ob-

Fig. 6.—Glass bell with linen bag containing calcium chloride, during measurement of insensible perspiration.

tained in two simultaneous measurements of symmetric areas of a subject's skin were within 5 per cent of equality.

The insensible perspiration of the patient with hypoplasia of sweat glands measured at complete rest in a lying position and at a room temperature of 73 F. was found to be 0.0214 Gm. I have found the insensible perspiration of normal persons under similar conditions to vary from 0.0188 to 0.0296 Gm. The patient's insensible perspiration thus falls within normal limits.

22. Kahn, D., and Rothman, S.: Sweat Response to Acetylcholine, J. Invest. Dermat. **5**:431, 1942.

23. Dale, H. H., and Feldberg, W.: The Chemical Transmission of Secretory Impulses to the Sweat Glands of the Cat, J. Physiol. **82**:121, 1934.

The injection of 0.5 mg. of atropine sulfate subcutaneously had no effect on the insensible perspiration of the patient when the measurement started fifteen minutes after injection of the drug.

COMMENT

The secretion of the apocrine glands has appeared normal in this patient, as well as in other patients with the anhidrotic type of ectodermal dysplasia.[8c] The function of eccrine glands was decreased to practically none on the face and abdomen. Still, approximately 5 to 7 per cent of the normal number of glands were present on the extremities. It is thus seen that in spite of hyperthermia and apparent clinical absence of sweat glands single glands may be present in persons with the so-called anhidrotic type of ectodermal dysplasia. Thus the term "hypohidrotic" would appear more correct than "anhidrotic." The distribution of these few glands was rather irregular in this patient.

It is generally agreed that water evaporating from the surface of the skin originates from two sources: first, from the active secretory function of the sweat glands and, second, from a passive transepidermal transport of water.[24] Sweat glands secrete water continuously, but at room temperature the amount is so slight that visible droplets are not formed. The microscopic droplets evaporate immediately after their appearance on the surface of the skin. The continuity of secretion at room temperature was proved by direct observation with the capillary microscope[25] and by the fact that the insensible perspiration may decrease after administration of atropine, which paralyzes function of the sweat glands. Moog[26] (1923) found values for perspiration decreased to 50 per cent, and Rothman[27] (1930) observed a variable decrease of 10 to 30 per cent in different persons after atropinization. Loewy and Wechselmann[28] (1911), working on anhidrotic patients, discovered their insensible perspiration to be normal at rest and at low temperatures. They also found atropine in doses of 1 mg. to have little or no effect on the insensible perspiration of either normal or anhidrotic persons. As stated previously, the present patient showed normal values for insensible perspiration not influenced by atropine. A healthy control subject also showed no change in insensible perspiration after a similar injection of 0.5 mg. of atropine sulfate subcutaneously. These observations indicate that the participation of sweat glands in insensible perspiration at room temperatures around 73 F. is rather small. It is probable that there are individual differences depending on the nervous excitability of the subjects tested.

SUMMARY

From detailed study of a patient with hereditary ectodermal dysplasia the following observations were made:

The family tree shows sex-linked inheritance, with male patients and female conductors.

Eccrine glands were practically absent on the face and abdomen. Their number on the extremities was decreased to 5 to 7 per cent of the normal number.

Apocrine sweat glands were abundant in the axillas, on the upper part of the chest and on the perimammillary areas.

The patient's insensible perspiration, at rest in a room temperature of 73 F., was normal. Atropine sulfate in a dose of 0.5 mg. subcutaneously did not change it.

The intradermal injection of a 1:100 solution of acetylcholine bromide produced only a few isolated spots of sweating on the patient's forearm, as contrasted with the decided response of a normal control subject.

In the heat chamber the body temperature of the patient rose rapidly, whereas the body temperature of normal persons did not change under similar conditions.

The term "hypohidrotic" is suggested as being more accurate than "anhidrotic" for the described type of ectodermal dysplasia. The absence of eccrine glands is not necessarily complete, in spite of hyperthermia at high external temperatures.

Dr. Stephen Rothman gave advice and direction in the study of this case, and Dr. J. R. Blayney cooperated in the dental examination.

950 East Fifty-Ninth Street.

24. Schwenkenbecher, A.: Die Haut als Exkretionsorgan, in Bethe, A.; von Bergmann, G.; Embden, G., and Ellinger, A.: Handbuch der normalen und pathologische Physiologie, Berlin, Julius Springer, 1929, vol. 4, p. 709.

25. Jürgensen, E.: Mikrobeobachtungen der Schweiss-Sekretion der Haut des Menschen unter Kontrastfärbung, Deutsches Arch. f. klin. Med. **144**:193, 1924.

26. Moog, O.: Der Einfluss von Pilocarpin, Atropin und Adrenalin auf die unmerkliche Hautwasserabgabe, Arch. f. exper. Path. u. Pharmakol. **98**:75, 1923.

27. Rothman, S.: Ueber die Wirkung des ultravioletten Lichtes auf die unmerkliche Wasserabgabe der Haut, Strahlentherapie **35**:381, 1930.

28. Loewy, A., and Wechselmann, W.: Zur Physiologie und Pathologie des Wasserwechsels und der Wärmeregulation seitens des Hautorganes, Virchows Arch. f. path. Anat. **206**:79, 1911. Loewy, A.: Untersuchungen über die physikalische Hautwasserabgabe, Biochem. Ztschr. **67**:242, 1914.

LOCALIZED MYXEDEMA IN ASSOCIATION WITH HYPERTHYROIDISM

JOSEPH C. AMERSBACH, M.D., AND BEN KANEE, M.D.
NEW YORK

Localized myxedema is comparatively rare in dermatologic practice. Attention was forcibly drawn to this fact when in checking the statistics of the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital for the past five years (1938 to 1942 inclusive), covering over 525,000 admissions, it was found that there was no case reported. Recently a patient has been admitted with localized myxedema of the tuberous variety associated with exophthalmic goiter. Since the cause of this disease is still unknown, with theories ranging from endocrine imbalance [1] to a more specific thyroid dysfunction,[2] it has been deemed advisable to report this case to stimulate interest in this dermatomedical problem.

REPORT OF A CASE

History.—H. W., a white man aged 30, a patient of Dr. John D. Currence, entered the New York Post-Graduate Medical School and Hospital on May 28, 1943 for roentgen therapy for recurrent hyperthyroidism, which had been recognized for the past four years. At the onset his basal metabolic rate was + 50 per cent. In 1940 he underwent a thyroidectomy at Dixon, Ill. Relief was transitory. In 1942 he underwent another thyroidectomy, at the Boston City Hospital. The thyroid gland promptly grew large again, after a brief period of relief. After the operation in 1940, he had five roentgen treatments, which, however, afforded him no relief. He had taken "bottles" of strong solution of iodine U. S. P. up to February 1943. At that time his basal metabolic rate was + 20 per cent. He entered the hospital because of obvious enlargement of the thyroid and continuation of symptoms, including a basal metabolic rate of + 48 per cent. He had lost 8 pounds (3.6 Kg.) since February. The patient had been aware of a nodular eruption on his legs for the last six months. His past history, other than that mentioned, was noncontributory.

Examination.—The patient was obese and apprehensive but not acutely ill. The skin was warm, moist and erythematous. The eyes showed a protrusion of the optic bulb and a slight lag of the lids. The pupils were equal and symmetric and reacted to light and in accommodation. The ears, nose and throat were essentially normal. The neck revealed a collar incision scar and a firm mass in the left thyroid region about 6 cm. long and 4 cm. wide. The lungs were clear to percussion and auscultation. The left border of the heart was 11 cm. from the midsternal line. A soft systolic murmur was audible at the apex. The heart sounds were of fair quality. The pulse rate when the patient was sitting was 118 per minute. There were no palpable masses in the abdomen. The reflexes were normal and equal on the two sides. The fingers showed a fine tremor.

There were numerous firm nodules about the anterior and lateral surface of the lower half of the legs and ankles, varying from 5 mm. to 2 cm. in diameter and elevated as much as 1.5 cm. The nodules did not pit on pressure, were not tender and appeared to be fixed to the skin but not attached to the underlying structures. The lesions were for the most part flesh colored; however, a few were erythematous. On the dorsa of the feet and toes and between the toes the skin was erythematous, exudative and scaly and the eruption clearly demarcated.

Laboratory Data.—The results of the examination of the blood were as follows: hemoglobin content, 14 Gm. per hundred cubic centimeters (84 per cent); red blood cells, 4,670,000 per cubic millimeter and white blood cells, 7,500, with polymorphonuclear neutrophils 70 per cent, polymorphonuclear eosinophils 3 per cent, lymphocytes 19 per cent, and monocytes 8 per cent. The sedimentation rate was 3 mm. per hour. Results of studies of the serum for bromide and iodine were as follows: bromide, less than 25 mg. of sodium bromide per hundred cubic centimeters; total iodine, 3 to 8 micrograms, and precipitable (organic) plasma iodine (a measure of thyroxin and diiodotyrosine), 11.3 micrograms per hundred cubic centimeters.

The basal metabolic rate was + 48 per cent.

A roentgenogram of the chest indicated neither gross cardiac nor pulmonary abnormality, and there was no evidence of thyroid extension.

Mycologic studies of scrapings and cultures of material from the toes did not reveal the presence of fungi.

Histologic Study.—A biopsy was performed and the histologic report by Dr. Charles F. Sims is as follows:

"Hematoxylin and Eosin Stain: The epidermis is moderately irregular. The surface is covered with a mild, loosely adherent horny layer. The granular layer is composed of from two to four layers of flattened rhomboidal cells. The rete reveals no apparent acanthosis. The basal margin is intact throughout.

"The corium reveals the following changes: In the upper portion some fragmentation of the collagen fibers may be noted. The collagen bundles present a parenchymatous edema throughout and take the acid dye lightly. Scattered throughout the corium are many stellate cells.

From the Skin and Cancer Unit, New York Post-Graduate Medical School and Hospital, Columbia University.

1. Lord, L. W., and Morrison, S.: Circumscribed Cutaneous Myxedema Associated with Possible Endocrine Imbalance, South. M. J. **26**:231-237 (March) 1933.

2. Schuerman, H.: Dermatoses and Thyroid Dysfunction, Arch. f. Dermat. u. Syph. **176**:544, 1938.

"Throughout the upper, middle and deep layers of the corium there is a substance which stains bluish to purplish and forms a network. Some of the purplish substance is composed of long and irregular masses or fibers, whereas others are short and broad. In the upper part of the corium, approaching the epidermis,

Fig. 1.—Exophthalmos and scar from previous thyroidectomies.

these purple-staining fibers become finer and more fragmented and give the appearance of fine granules.

"The vessels of the upper part of the corium are moderately dilated; their walls are somewhat edematous and are surrounded by a sparse cellular infiltration, composed for the most part of small round cells.

"Thionine Stain for Mucin: The section was stained with thionine for mucin in the following manner: The tissue was fixed in absolute alcohol and embedded in paraffin. The section was then stained for thirty minutes in a 1 per cent solution of thionine in 25 per cent alcohol. The section was differentiated in 95 per cent alcohol followed by absolute alcohol, cleared in xylene and mounted in balsam. The mucin appeared light red to dark red or purple, depending on the intensity of the stain.

"The purplish or bluish substance seen in the section stained with hematoxylin and eosin appeared as a dark red to purplish network when stained with thionine, whereas the normal collagen remained unstained."

Treatment and Subsequent Course.—Filtered roentgen rays were applied to the area of the thyroid gland. During the patient's stay in the hospital, there seemed to be some regression of the myxedematous lesions.

COMMENT

In cases of localized myxedema of the lower extremities associated with exophthalmic goiter studied by O'Leary,[3] all of the patients showed auricular fibrillation and edema of the extremities. Our patient as yet has presented no evidence of serious cardiac dysfunction or decompensation; however, since the hyperthyroidism has not been ameliorated by standard therapy over a period of four years, the prognosis must remain guarded.

Pillsbury and Stokes[4] in a review of the literature cited 23 cases of circumscribed myxedema of the skin. They noted that in all but 1 of the 10 cases of the nodular variety it was associated with diffuse myxedematous infiltrations of other portions of the body, particularly the face. They also noted that in 12 of the 23 cases it was associated with hyperthyroidism and edema of the legs. It should be noted that in our case, in

Fig. 2.—Tuberous condition of the legs.

3. O'Leary, P. A.: Localized Solid Edema of the Extremities in Association with Exophthalmic Goiter, Arch. Dermat. & Syph. **21**:57 (Jan.) 1940.

4. Pillsbury, D. M., and Stokes, J. H.: Circumscribed Myxedema of the Skin, Arch. Dermat. & Syph. **24**:255 (Aug.) 1931.

which the myxedema is of the tuberous or nodular variety the eruption was limited solely to the patient's lower extremities.

Many theories have been advanced to explain the involvement of the lower extremities. Pillsbury and Stokes [4] expressed the opinion that circulatory stasis plays an important role, and Stryker [5] suggested that trauma is a possible factor.

In a differential diagnosis of the eruption on the dorsa of the feet and toes, dermatitis venenata and dermatophytosis had to be considered. The eruption on the legs and ankles suggested the following possibilities clinically: (1) erythema-nodosum-like dermatitis medicamentosa due to iodides or bromides, (2) thrombophlebitis and (3) localized myxedema.

5. Stryker, in discussion on Lord and Morrison.[3]

SUMMARY

Localized myxedema of the tuberous variety developed in a patient who had been suffering from exophthalmic goiter for four years and who had persistent hyperthyroidism despite protracted therapy, including two thyroidectomies.

Histologic studies showed changes in the cutis typical of myxedema.

Dr. John D. Currence gave permission to report this case.

105 East Fifty-Third Street.

Vancouver, British Columbia, Canada.

FIXED ERYTHEMA DUE TO SULFANILAMIDE WITH GRADUALLY LESSENING SENSITIVITY

A. DOSTROVSKI, M.D., AND F. SAGHER, M.D.
JERUSALEM, PALESTINE

Sulfanilamide and its derivatives are now being so widely employed in practical therapy that any symptom occurring as a result of the administration of that drug should receive due attention; this is particularly true of the so-called "fixed erythema." In the literature reference has been made to this eruption by Loveman and Simon [1] and later by Goodman and Arthur.[2] In both reports definitely localized eruptions on various parts of the patients' bodies were described, in which renewed administration of the drug provoked the same reactions, but by both authors a certain tendency of the eruption to involve other parts of the healthy skin too has been noted.

The case herein reported shows some peculiarities which have not been encountered in the others: The localization was invariably the same; the eruptions greatly resembled those called forth by phenolphthalein, and after the lapse of a year a definite decline of sensitivity could be noted.

REPORT OF A CASE

The patient was 34 years old. One of his brothers was suffering from hay fever, but the family history otherwise presented nothing abnormal. The patient had urticaria when he was 13. Of later diseases he reported that he had had typhus, catarrhal jaundice and repeatedly recurring tonsillitis. Six months before being seen he had such an attack of tonsillitis, and at about the same time he noticed that small vesicles had formed on the skin of his chest, similar in appearance to a herpes eruption (according to the patient, who is a physician himself); this later healed with pigmentation. Only then did he take sulfanilamide (0.5 Gm.), but up to the following attack of tonsillitis he took nothing.

When we saw him, he had had 1 Gm. of sulfanilamide two days earlier on account of another attack of tonsillitis, and on the following morning he noticed circumscribed redness and swelling of the skin exactly covering the pigmented area. Simultaneously with sulfanilamide he had taken opium and activated charcoal.

Examination.—May 18, 1942: Slightly beneath a line running from the sternal border to the left nipple there was an oval swollen area, 5 by 3 cm. Three separate zones were clearly distinguishable: (1) in the center, a small red circle; (2) a pigmented circle surrounding the former, and (3) an outer circle where the skin was red and swollen. The whole lesion was elevated above the level of the skin in plateau fashion. After the lapse of twenty-four hours the swelling had to some degree subsided, and after another three days it had completely disappeared. The pigmented circle, however, remained clearly recognizable all the time.

Although it was without doubt a fixed erythema, the following experiments were undertaken to corroborate the diagnosis. (Photographs were taken after administration of the drug and after the reaction had disappeared.)

June 28: The patient was given 0.5 Gm. of sulfanilamide. Five hours later he reported slight itching and after eight hours swelling of the skin was noted (fig. *A*). A red halo surrounded the central pigmentation in an area exactly coinciding with that involved six weeks earlier. After five days the symptoms were still rather intense, but after eight days they had disappeared, leaving a circular pigmentation (fig. *B* [taken six months later]). Patch tests with sulfanilamide on the intact skin of the arm as well as a scratch test gave negative results.

July 15: The patient received 0.5 Gm. of sulfapyridine, but no reaction developed.

August 4: This time he was given 0.5 Gm. of a brand of sulfanilamide manufactured in Palestine (albosal). The reaction that occurred was of similar intensity to that noted on the first occasion. After two days the symptoms were still recognizable, but they disappeared somewhat more rapidly than in the first experiment.

To establish the minimum dose with which the reaction would be provoked, the patient was given 0.005 Gm. of sulfanilamide; no reaction followed.

August 31: The patient was given 0.045 Gm. of sulfanilamide. After eight hours the intensity of the reaction had already reached its peak, but after twelve hours there was only a slightly red circular band 1 to 2 mm. wide surrounding the central pigmentation. A blood count was normal.

September 25: The patient was given 1.5 Gm. of sulfapyridine, with no reaction.

October 8: One gram of sulfanilamide was given by intramuscular injection; no reaction followed.

October 15: This time 0.5 Gm. of sulfanilamide provoked a mild but distinct reaction, which disappeared after forty-eight hours.

December 26: This time, two months later, the patient was again given 0.5 Gm. of sulfanilamide per os, with no reaction.

December 29: One gram of sulfanilamide was given per os; there followed a scarcely recognizable redness of the skin.

From the Dermatologic Department of the Rothschild-Hadassah University Hospital.

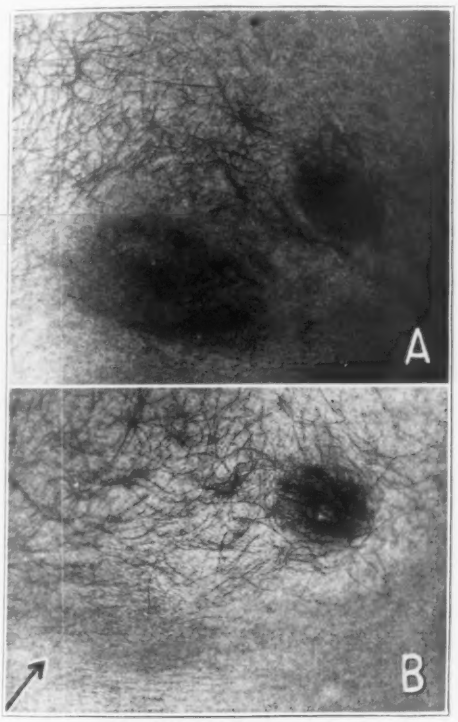
1. Loveman, A. B., and Simon, F. A.: Fixed Eruption and Stomatitis Due to Sulfanilamide, Arch. Dermat. & Syph. **40**:29 (July) 1939.

2. Goodman, M. H., and Arthur, R. D.: Fixed Eruptions: Report of an Unusual Condition Due to Sulfanilamide, Arch. Dermat. & Syph. **43**:692 (April) 1941.

Jan. 25, 1943: A scratch test with sulfanilamide in the affected area elicited a negative reaction.

April 14: One gram of sulfanilamide per os was followed by scarcely recognizable redness of the skin.

An otherwise normal patient who was suffering from recurring attacks of tonsillitis suddenly on the occasion of one of his usual attacks noticed on the skin of his chest a circumscribed patch studded with small vesicles which after a while disappeared and was followed by pigmentation. It was only later that he took sulfanilamide. When sulfanilamide was given again, after half a year, redness and swelling developed on exactly the same spot of the skin. Attempts to reproduce this fixed erythema artificially were successful seven times. The minimum dose required for its provocation was 0.045 Gm. of sulfanilamide. During nearly one full year this reaction appeared but with gradually lessening intensity, so that it was eventually scarcely recognizable even after administration of 1 Gm. of the drug. This must be considered a consequence of desensitization which had taken place in the meantime either spontaneously or induced by the repeated test administrations of the same drug. Neither intramuscular injections of sulfanilamide nor administration of sulfapyridine caused a similar eruption. Patch tests on the normal skin as well as on the affected area elicited negative reactions.



A, fixed erythema due to sulfanilamide. In the center of the erythematourticarial swelling is a pigmented circle, the residue of an earlier inflammation. *B*, appearance six months later. Only the slightly pigmented plaque is still recognizable.

COMMENT

While in the literature disseminated fixed foci are reported on—in 1 case involving the mucous membrane of the lips too—our patient had only one isolated plaque. In a pigmented area predisposed by earlier inflammation (perhaps herpes) the administration of sulfanilamide provoked the appearance of a fixed erythema. Since the symptoms were similar to those produced by phenolphthalein, the sulfanilamide preparation which has been used was tested (Professor Wertheimer) for the possible presence of traces of that drug, but with a negative result. Strikingly in our case, as well as in the others referred to in the literature, the symptoms occurred only after administration of sulfanilamide and not after use of sulfapyridine. Azosulfamide, which was for the sake of comparison tried by Loveman and Simon,[1] provoked the same sort of reaction, although of much less intensity. Since in azosulfamide far smaller amounts of paraaminobenzenesulfonamide are released, it is apparently the presence of this complex that accounts for the appearance of the symptoms.

That patch tests elicited negative reactions was not particularly surprising, since this is the rule in all kinds of fixed erythemas. When, however, they tested the affected area itself, Loveman and Simon in 1 instance were able to provoke a mild erythema there. In our case the drug was applied to the affected area only at a time when desensitization was already far advanced; hence the result was naturally negative. The sulfonamide compounds, moreover, elicit positive reactions to patch tests only in the presence of eczematous changes of the skin, as, for example, in the case reported on by Myers, Vonder Heide and Balcerski.[3] On intramuscular injection the results were negative, as has similarly been pointed out by Abramowitz [4] in his article dealing with phenolphthalein, the barbiturates and arsphenamine.

As to the minimum dose required to provoke the cutaneous reaction, it was noteworthy that one one-hundredth of the dose taken by our patient produced no effect, while on administration of one-tenth the dose the symptoms were pronounced. This is, however, generally the case with fixed erythemas of all kinds, and it is known

3. Myers, G. B.; Vonder Heide, E. C., and Balcerski, M.: Exfoliative Dermatitis Following Sulfanilamide, J. A. M. A. **109**:1983 (Dec. 11) 1937.

4. Abramowitz, E. W.: Fixed Eruptions from Various Drugs and Other Agents, Arch. Dermat. & Syph. **43**:672 (April) 1941.

that they can be produced by doses which otherwise—therapeutically as well as toxicologically—are absolutely ineffective.

Passive transmission or experimental production of fixed erythema in man as well as in animals has so far not been possible; hence we refrain from touching on this subject.

It is particularly interesting that the reaction gradually declined in intensity. This fact, as well as an intensification or a polyvalent character that the sensitivity may occasionally assume, is known and has repeatedly been pointed out. However, as to sulfanilamide itself nothing of the kind has been referred to in earlier publications. In our case the occurrence of fixed erythema was recorded—with greater or lesser intensity proportionate to the dose employed—seven times in the course of ten months. After the lapse of six months the dose of 1 Gm. was able to provoke scarcely any reaction at all. This desensitization may have been spontaneous, it may, however, at least in some measure have been the result of the repeated experimental administrations of the drug.

SUMMARY

In the case of a patient under our care the administration of sulfanilamide provoked the appearance of fixed erythema in a predisposed area of the skin. During the course of ten months the intensity of the reaction was gradually reduced.

COAL TAR IN DERMATOLOGIC PREPARATIONS

J. G. DOWNING, M.D.; L. M. OHMART, A.M.,*
AND G. DI CICCO, B.S.*
BOSTON

Crude coal tar is probably one of the most efficient and useful substances employed in the practice of dermatology. Many dermatologists have insisted that their prescriptions for ointments and pastes containing coal tar be dispensed with a black or nearly black color. If this color was not obtained, these practitioners believed that the expected therapeutic results would not be obtained. As a result, by custom and tradition, it has been the practice of many dermatologists in prescribing these formulas to refer their patients to pharmacists who dispense ointments and pastes containing coal tar and zinc oxide with a black or nearly black color. The black color is obtained by mixing the zinc oxide with the coal tar before incorporating any of the base. This procedure is contrary to accepted pharmaceutic technic and usually results in a rather gritty preparation, since there is no opportunity for proper levigation of the zinc oxide. The normal mode of procedure would be to levigate the zinc oxide with a portion of the base and then add successively the remainder of the base, the starch and finally the coal tar. This method produces a gray or gray-green product, depending on the nature of the coal tar used. The texture is superior to that of the black product, there being no grittiness due to incomplete levigation of the zinc oxide.

Since some dermatologists appear to be of the opinion that coal tar preparations which are black are therapeutically superior to those which are gray, an investigation was undertaken to establish their relative merits.

EXPERIMENTAL PROCEDURE

Using the following formula as a prototype of coal tar ointments and pastes in common use, we prepared two series of samples.

	Gm.
Crude coal tar	2.0
Zinc oxide	2.0
Starch	15.0
Petrolatum	15.0

Those of series A were prepared by levigating the zinc oxide with the coal tar, adding the petrolatum and finally the starch. Those of series B were prepared by levigating the zinc oxide with a portion of the petrolatum, adding the remainder of the petrolatum, then the starch and finally the coal tar.

These samples were then tested clinically. The experimental subjects selected were 30 workers suffering from an occupational dermatitis. The pastes were applied to the skin of the patients suffering from a dermatitis of both hands. The traditional black formula, series A, was applied to the skin of one hand and the gray mixture, series B, to the other. The results were fairly uniform, there being no significant difference in the response to the two preparations.

It is our opinion that the preference of many dermatologists for coal tar preparations which are black may have had its origin in the variability of market samples of coal tar. This variation and the skill of the compounding pharmacist are the important factors, not the color.

One of the disadvantages encountered in the use of ointments and pastes containing coal tar lies in the difficulty of their removal from the body. Removal is facilitated by use of olive oil or some other fixed oil, but this method has its own obvious disadvantages. Even with the aid of a fixed oil, removal is not easy from a body surface which is hairy or where the skin is not intact.

Since the physical nature of petrolatum appears to be the cause of the difficulty, a special base was prepared and used in place of the usual petrolatum in the following preparation.

	Gm.
Crude coal tar	2.0
Zinc oxide	2.0
Starch	15.0
Base	15.0

* From the Massachusetts College of Pharmacy, Department of Pharmacy.

The special base contained 5 per cent of diglycol stearate and 95 per cent of petrolatum and was prepared by fusing these two substances together and stirring the product until it congealed.

The coal tar ointment prepared in this manner is easily removed from body surfaces with dry tissues or fabrics or by the use of soap and water.

SUMMARY

Many dermatologists are of the opinion that ointments and pastes containing coal tar and zinc oxide which are black are therapeutically superior to preparations which are gray. The black color is developed by a method of compounding which is contrary to accepted pharmaceutic technic and which results in a somewhat gritty preparation.

In order to determine the relative therapeutic merits of the black and the gray products, two series of samples of a coal tar and zinc oxide paste were prepared. The samples were tested clinically. No significant differences were observed in the therapeutic effects.

Coal tar ointments and pastes are difficult to remove from the surface of the body. Removal is facilitated by using, instead of the usual petrolatum, a base consisting of 5 per cent diglycol stearate and 95 per cent petrolatum.

BULLOUS LICHEN SCLEROSUS ET ATROPHICUS

ITS RELATION TO BULLOUS SCLERODERMA

C. RUSSELL ANDERSON, M.D.

LOS ANGELES

In 1940 Montgomrey and Hill [1] in their classic article on lichen sclerosus et atrophicus reviewed the clinical and histologic features of this disease. Their observations were based on a study of 46 cases. They found that confusion had probably resulted, first, because of the close similarity of the cutaneous pictures of lichen sclerosus et atrophicus and some examples of morphea guttata and, secondly, because a histologic picture for lichen sclerosus et atrophicus distinct from that of morphea and from that of atrophic lichen planus had not generally been recognized.

In 2 of their cases there were infiltrative plaques suggestive of local scleroderma. In 1 of their cases there were multiple infiltrated plaques simulating those of an edematous stage of morphea but with characteristic keratotic plugs clinically and histologically.

The clinical features of lichen sclerosus et atrophicus [1] are so well known that they do not require repetition. It is sufficient to recall that the individual papule, whether discrete or as one of an aggregation forming a plaque, has on its surface one or more dark horny comedo-like plugs or minute beadlike depressions, which show the former sites of horny plugs. Montgomery and Hill [1] stated that especially in the early stages of the disease keratotic plugs or depressions may not be recognized clinically but are almost invariably present on pathologic examination. In the stage of involution a white parchment-like or tissue-paper-like wrinkling may be seen.

These authors found the characteristic histologic changes of lichen sclerosus et atrophicus to consist of relative and absolute hyperkeratosis, with keratotic plugging of the hair follicles and dermal appendages, and also, independent of either, atrophy of the rest of the epidermis, flattening and loss of the rete ridges, mild liquefaction degeneration or necrosis of the basal cell layer, which is the result of dilatation of the lymphatic spaces and vessels, lymphedema in the upper portion of the cutis, homogenization and edematous changes in the connective tissue fibers beneath the epidermis, separation of elastic tissue fibers from the epidermis but without their destruction, a variable infiltrate in the midcutis beneath the area of edema and absence of obliterative changes in the deeper blood vessels. They stated that the edema in the upper part of the cutis may be so great as to result clinically in the formation of small vesicles, and they referred to the cases reported by Myers and Schubert.

Myers' [2] patient was a 65 year old woman who had multiple lesions of lichen sclerosus et atrophicus of the back. In two of the plaques in the lumbosacral region flat bullae with thick horny roofs were present. Fluid was obtained from the bullae. Histologic examination revealed a picture characteristic of lichen sclerosus et atrophicus.

Schubert's [3] patient was a 15 year old girl who presented a typical eruption of lichen sclerosus et atrophicus. Some of the patches had a finely folded, somewhat movable epidermis with individual small yellowish gray horny plugs and a palpable elevation above the surrounding skin. After insertion of a needle, a drop of clear yellow fluid was obtained. The folded, thin epidermis could be easily scratched off, and this was followed by weeping and crust formation. As an end result, after healing, a number of small round smooth whitish scars with fine telangiectases over the atrophic epidermis were left. The clinical illustration (fig. 3) of Schubert's article is a counterpart of the clinical illustration (fig. 1) of Templeton's article on localized scleroderma with bullae.[4]

The histologic illustrations in Schubert's article correspond in every way with those in Montgomery and Hill's article [1] and in Templeton's article.[4]

---

1. Montgomery, H., and Hill, W. R.: Lichen Sclerosus et Atrophicus, Arch. Dermat. & Syph. **42**:755-779 (Nov.) 1940.

2. Myers, W. K.: Lichen Sclerosus: Case Report, Brit. J. Dermat. **48**:658-660 (Dec.) 1936.

3. Schubert, M.: Zur Kenntnis des Lichen sclerosus atrophicans, Dermat. Wchnschr. **103**:1653-1662 (Dec. 19) 1936.

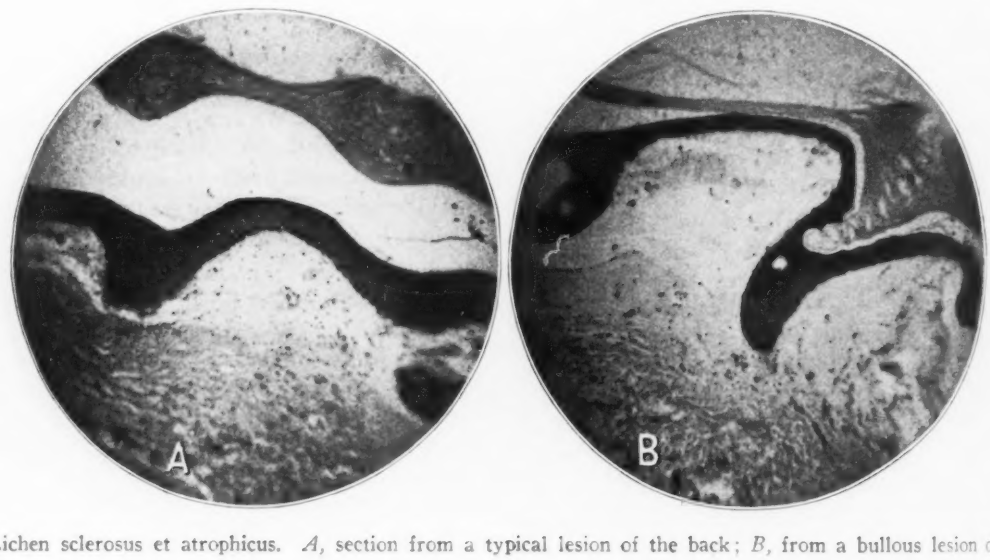
4. Templeton, H. J.: Localized Scleroderma with Bullae, Arch. Dermat. & Syph. **43**:361-365 (Feb.) 1941.

REPORT OF A CASE

Mrs. R. H., aged 65, was seen in consultation on Oct. 13, 1941. She stated that for five years white spots had been appearing over her body. There had been no subjective symptoms except on the lower part of the back and on the genitalia, where itching and stinging sensations were present. She had noted recently that blisters had appeared in some of the white spots under the breasts and on the abdomen. She had used no local therapy.

On examination a widespread eruption of lichen sclerosus et atrophicus, involving the trunk, arms, groins and genitalia, was found. The most extensive involvement was over the shoulders anteriorly and from the clavicles to the lower part of the abdomen. On the abdomen and under the breasts several of the patches had a superficial wrinkled appearance, the wrinkles running in the direction of the lines of cleavage. On palpation this appeared to be due to the presence of an atrophic epidermis overlying a collection of fluid. There were also several patches where small, pinhead-sized to matchhead-sized, vesicles were present. In the midlumbar region there was a half-dollar-sized patch of lichen sclerosus with a violaceous areola and, in addition, a flat bullous lesion. In the lower midlumbar region were three silver dollar–sized plaques with a wrinkled epidermis and vesicles and flat bullae. Puncture of the vesicles and bullae yielded a clear yellowish fluid.

Biopsy was performed on a typical patch of lichen sclerosus et atrophicus of the upper part of the back and on two bullous lesions of the midlumbar region. Histologic examination (the figure) showed all the characteristic findings of lichen sclerosus et atrophicus as described by Montgomery and Hill and so require no repetition. The figure may be compared with the histologic illustrations in Montgomery and Hill's paper and with the histologic illustrations in Templeton's paper.



Lichen sclerosus et atrophicus. *A*, section from a typical lesion of the back; *B*, from a bullous lesion of the back.

LOCALIZED SCLERODERMA WITH BULLAE

From the older literature Luithlen [5] mentioned cases of Brummond, Hallopeau, Hardy, Bouttier, Morrow, Leisrink and Lepine in which vesicles formed in localized scleroderma. Full details could be obtained only about the case of Morrow.[6] His patient was a man aged 66 with extensive generalized symmetric morphea attended with the formation of bullae followed by ulceration and scarring. The illustrations accompanying his paper clearly show the keratotic plugging of morphea-like plaques. He also wrote that "the white central areas are stippled over with numerous punctiform indentations, corresponding doubtless to the follicles of the skin." Morrow's case apparently was one of lichen sclerosus et atrophicus.

Pautrier's [7] patient was a woman aged 28 who presented a large number of areas of circumscribed scleroderma over the whole body. In some of these plaques vesicle formation followed by ulceration occurred. The illustrations were not clear, and the histologic description was inadequate.

---

5. Luithlen, F.: Sclerodermie, in Mracek, F.: Handbuch der Hautkrankheiten, Vienna, A. Holder, 1904, vol. 3, pp. 128-192.

6. Morrow, P. A.: A Case of Symmetrical Morphea Attended with the Formation of Bullae and Extensive Ulceration, J. Genito-Urin. Dis. **14**:419-428 (Nov.) 1896.

7. Pautrier, M. L.: Sclérodermie à évolution rapide en plaques multiple, Bull. Soc. franç. de dermat. et syph. **36**:928-938 (May 26) 1929.

Jadassohn [8] cited Freund as having observed a case of typical *sclérodermie en plaques* with clearly defined lilac rings which showed a bleb-like lifting of the epidermis in the middle of the scleroderматous area. I was unable to find the original description of this patient.

In a personal communication to Templeton,[4] O'Leary wrote of a woman under his care who had a generalized morphea in the sense that she had a multitude of sclerodermatous plaques over the abdomen and legs in which bullae developed. He considered the bullae to be the result of trauma. In a later personal communication [9] to me concerning the same patient, O'Leary stated that she now presented superficial cigaret-paper-like atrophy of the chest and back and that she had some white spot disease remaining on the back. Histologic study had not been made.

R. L. Sutton [10] wrote that he had had 1 patient with morphea with vesiculation under his care. He gave no clinical or histologic details.

Templeton's [4] patient was a woman aged 23. She presented coin-sized lesions on the back, chest and abdomen of five years' duration. The lesions were apparently in different stages of evolution. Three on her chest, which were said to be the oldest, represented the end stage of some atrophic process, as the skin was depigmented and definitely thinned. On the right scapula there was a lesion about 3 cm. in diameter which was atrophic, depigmented and slightly scaly and showed some telangiectases. On the lower part of the abdomen there was an area measuring 5 by 8 cm. which was rather typical of the edematous stage of scleroderma, being indurated and ivory-colored with a lilac border. Two months later different stages of a bullous reaction could be seen in some of the lesions (figure 1 of Templeton's article). The most typical bulla occupied the center of the indurated ivory-colored sclerodermatous lesion on the abdomen. The walls of the bulla were thick, and the material in it was somewhat gelatinous and semiorganized. Templeton observed this patient for five years. A few new sclerodermatous patches appeared at times. The lesions in time became atrophic; bullae became superimposed on some of them, and when they did so the bullae persisted for many weeks, slowly turning into crusts and finally falling off. His report of the histopathologic examination showed the epidermis to be thinned, with pronounced diminution and absence of the interpapillary prolongations of the rete. The stratum corneum showed changes consisting of splitting and in certain areas fairly pronounced subcorneal bleb formation. The stratum squamosum was noticeably thinned. A slight amount of cellular edema was noted in the deeper layer of the cells near and in the actual basal layer. In the corium two striking changes were evident; edema and lymphatic dilatation in the pars papillaris and definite sclerodermatous thickening of the collagen in the subpapillary and reticular layers. The edema of the papillary layer was apparently cellular, as the collagen fibers were swollen and had lost their affinity for the acid stain. In some areas they were a pale almost homogeneous mass. All degrees of dilatation of the lymphatics, from slightly dilated vessels to huge spaces, were present in the papillary and subpapillary layers. In some areas the pronounced edema of the papillary layer and the decided dilatation of the lymph vessels combined to form huge lacunar spaces, which were apparently the blebs that were seen clinically. The reticular layer was considerably thickened and was occupied by bundles of collagen which showed a moderate degree of swelling, such as is seen in ordinary scleroderma. There was no noteworthy invasion of the corium by lymphocytes, nor was there any decided perivascular infiltrate. The vascular changes consisted mainly of the pronounced dilatation of the lymphatics in the upper layers of the corium.

However, on close examination of the illustrations accompanying Templeton's article, certain clinical and pathologic features are noted that were not mentioned by him. His clinical illustrations (Templeton's figure 1) show the plication, or accordion-pleated surface, often seen in lichen sclerosus et atrophicus. Also all the clinical illustrations clearly show the keratotic plugging characteristic of lichen sclerosus et atrophicus. The first histologic illustration shows the subpapillary lymphocytic infiltrate. The histologic illustration (Templeton's figure 2 *B*) also shows the relative and absolute hyperkeratosis and the striking keratotic plugging of the epidermis, in relation to the pilosebaceous orifices and also independent thereof. These changes are not seen in localized scleroderma or morphea, but they are constantly seen in lichen sclerosus et atrophicus. Montgomery and Hill stated that in a large series of cases of morphea, including many of morphea guttata, studied pathologically at the

8. Jadassohn, J.: Handbuch der Haut- und Geschlectskrankheiten, Berlin, Julius Springer, 1931, vol. 8, pt. 2, pp. 728-729.

9. O'Leary, P. A.: Personal communication to the author.

10. Sutton, R. L., and Sutton, R. L., Jr.: Diseases of the Skin, ed. 10, St. Louis, C. V. Mosby Company, 1939, p. 484.

Mayo Clinic they had failed to observe keratotic plugging such as is constantly seen in lichen sclerosus. The histologic picture as shown in Templeton's article is practically identical with the histologic picture of lichen sclerosus et atrophicus as shown in figure 6 *A* in the article by Montgomery and Hill.[1]

I believe that the case reported by Dr. Templeton as a case of bullous localized scleroderma must be considered to be one of bullous lichen sclerosus et atrophicus. All the criteria for the histologic diagnosis of lichen sclerosus as described by Montgomery and Hill are present in his case.

The complete differential diagnosis of lichen sclerosus et atrophicus will not be given here, for reference may be made to the article by Montgomery and Hill.[1]

SUMMARY AND CONCLUSIONS

It is not denied that there is such an entity as localized scleroderma with bullous lesions; however, at the present time there is no histologic evidence to support such a diagnosis. It is probable that most of the cases reported have been, instead, cases of lichen sclerosus et atrophicus with bullous lesions.

1930 Wilshire Boulevard.

CUTANEOUS ABSORPTION

II. THE VALUE OF PETROLATUM, ANHYDROUS WOOL FAT AND OTHER BASES IN PERCUTANEOUS ABSORPTION OF TOPICALLY APPLIED COTTONSEED ALLERGEN

ABRAHAM WALZER, M.D., AND SAMUEL S. SACK, M.D.
BROOKLYN

In a previous communication [1] it was demonstrated that proteins, such as cottonseed, when applied topically are absorbed through the skin of human beings and of monkeys. In that study a site on the skin of the subject to be tested was passively sensitized to cottonseed antigen. After twenty-four to forty-eight hours, the antigen, suspended in a grease base, was rubbed in at another cutaneous site. The development of a wheal at the sensitized site marked the entrance of detectable amounts of antigen into the circulation.

The serum for sensitization was obtained from a patient strongly sensitive to cottonseed and with an extremely high titer of reagins for this antigen (1:512 by the dilution method of Coca and Grove [2]). The serum was used in a dilution of 1:4 in human subjects and undiluted in monkeys. From 0.05 to 0.1 cc. was injected intracutaneously for local passive sensitization.

The antigen inunctum was prepared in the following manner: One or 2 Gm. of dehulled cottonseed was vigorously triturated until it was reduced to a gummy mass. An equal amount of a base, such as petrolatum, anhydrous wool fat or a similar substance, was then added and thoroughly mixed with the cottonseed. Four grams of this antigen ointment was used for each inunction in human subjects and 2 Gm. in monkeys.

In the studies on the Macacus rhesus monkeys, corresponding areas on opposite sides of the body were carefully shaved. One of these was employed for sensitization and the other for inunction of the antigen.

The reaction in the human subjects began at the sensitized site from eleven to fifty-one minutes after the onset of inunction. It started with an itch and faint erythema and was followed by development of a wheal within the erythematous area.[1] The reaction in the monkeys usually appeared at the sensitized site in from five to sixteen minutes after the rubbing was begun. The most common type of reaction began with the development of pinpoint-sized elevated points at the hair follicles. Within a minute or two the affected area became ivory white and slightly elevated, but no areola accompanied these reactions. Detailed descriptions of the numerous variations in cutaneous reactions encountered in the monkeys were given in the previous communication.[1]

In the present investigation the comparative rates of percutaneous absorption of cottonseed suspended in various vehicles were studied. Most of the experiments were performed with petrolatum and anhydrous wool fat. Preliminary studies were also started with other vehicles, but because of unavoidable circumstances these investigations had to be discontinued. It was considered advisable, nevertheless, to present at this time all of the results now available.

The technic employed in these experiments did not vary from that described in the previous communication [1] and summarized in the preceding paragraphs. When tests were repeated on a subject, an interval of one or two weeks was allowed to elapse between studies to allow time for the complete elimination of antigen from the circulation [3]

The vehicles employed in the present studies were petrolatum, anhydrous wool fat, oil of anise, cetyl alcohol and petrolatum with an alkali.

RESULTS

1. Petrolatum.—Human Subjects: Thirty-four adult subjects were tested (table 1). In 7, or 20.6 per cent of these, no reaction developed at the sensitized site, which indicates a failure to absorb the antigen. Two showed greatly delayed

From the Department of Dermatology and Syphilology and the Department of Allergy of the Jewish Hospital of Brooklyn.

1. Walzer, A.: Cutaneous Absorption: I. A Direct Technic for Demonstrating the Percutaneous Absorption of Antigens, Arch. Dermat. & Syph. **41**:692 (April) 1940.

2. Coca, A. F., and Grove, E. F.: Studies in Hypersensitiveness: XIII. A Study of the Atopic Reagins, J. Immunol. **10**:445, 1925.

3. Walzer, A., and Walzer, M.: Studies in Absorption of Undigested Proteins in Human Beings: V. A New Technic for Quantitatively Studying the Absorption and Elimination of Antigens (Preliminary Report), J. Allergy **6**:532, 1935.

reactions, eighty-seven and one hundred and sixty-five minutes. Of the remaining 25, the reacting times at the sensitized sites varied from eleven to fifty-one minutes after the onset of the inunction. Among these 25 subjects, 12 reacted in from eleven to nineteen minutes, 6 in from twenty to twenty-four minutes, 4 in from thirty to thirty-four minutes and the remaining 3 in forty-five, forty-seven and fifty-one minutes respectively. The average time required for the entrance of antigen into the blood in these 25 subjects was twenty-three and two-tenths minutes after the onset of the inunction of the cottonseed ointment.

TABLE 1.—*Absorption Rates in Human Subjects for Cottonseed Suspended in Various Bases*

Subject No.	Time of Appearance of Reaction, Minutes: Petrolatum	Anhydrous Wool Fat	Cetyl Alcohol	Oil of Anise
1	24	31	..	..
2	51	..	..	..
3	31	..	..	..
4	18	15	..	..
5	Negative	..	..	..
6	15	..	..	..
7	Negative	..	..	..
8	13	..	..	..
9	20	30	..	..
10	23	17	..	..
11	Negative	Negative	..	..
12	20	..	..	..
13	Negative	..	..	..
14	11	13	..	..
15	18	..	..	..
16	87*	..	..	..
17	24	..	..	..
18	165*	..	..	..
19	Negative	..	..	..
20	Negative	..	..	..
21	45	..	..	..
22	Negative	..	..	..
23	30	Negative	12 hours *	..
24	19	11	..	..
25	31	..	..	..
26	34	Negative	..	..
27	47	..	..	..
28	..	32	51	..
29	..	20	Negative	..
30	..	24	Negative	..
31	..	21	34	..
32	..	20.5 hours *	36 hours *	..
33	..	25	42	..
34	20	23	..	23
35	14	17	..	..
36	12	11	..	11
37	15	18	..	..
38	19	15	..	..
39	13	..	..	..
40	12	12	..	..

* These readings were made by the subjects.

Monkeys as Subjects: Absorption tests with petrolatum as a base were made on 14 monkeys (table 2). One failed to react. Of the remaining 13, 7 showed evidences of absorption in from five to nine minutes and 6 in from ten to sixteen minutes. The average for this series of animals was nine and eight-tenths minutes.

II. Anhydrous Wool Fat.—Human Subjects: There were 21 subjects on whom the absorption of cottonseed suspended in anhydrous wool fat was studied (table 1). Three, or 14.3 per cent, failed to react. One reacted, according to his own observation, in twenty and five-tenths hours. The absorption times of the remaining 17 subjects ranged from eleven to thirty-two minutes. Reactions occurred in 9 in from eleven to eighteen minutes, in 5 in from twenty to twenty-five minutes and in 3 in from thirty to thirty-two minutes. In the 17 subjects the average time for the absorption of the cottonseed antigen suspended in anhydrous wool fat was nineteen and seven-tenths minutes.

Monkeys as Subjects: Only 3 monkeys were studied with inunctions of cottonseed in anhydrous wool fat (table 2). The reactions occurred in five, seven and five-tenths and nine minutes respectively, with an average of seven and two-tenths minutes for the group.

III. Oil of Anise.—Human Subjects: Before the interruption of the experiments, oil of anise was employed as an inunction base for cottonseed in tests on 2 subjects (table 1). The reactions in these 2 subjects appeared in eleven and twenty-three minutes respectively.

TABLE 2.—*Absorption Rates in Monkeys for Cottonseed Suspended in Various Bases*

Monkey No.	Time of Appearance of Reaction, Minutes: Petrolatum	Anhydrous Wool Fat	Oil of Anise	Petrolatum with 1% Potassium Carbonate
34	16	..	..	10
17	10	..	..	..
19	11	..	..	5
53	8	..	..	5
38	6	..	..	10
36	7	..	..	5.5
23	5	..	..	..
28	7	..	..	..
15	Negative	..	..	Negative
7	14	..	10.5	..
10	8	..	8	..
16	11	7.5	11	..
18	9	5	8	..
2	16	9	..	..

Monkeys as Subjects: Tests with oil of anise as a base were performed on 4 monkeys (table 2). Reactions occurred in eight, eight, ten and five-tenths and eleven minutes respectively, with an average for the group of nine and four tenths minutes.

IV. Cetyl Alcohol.—Human Subjects: Cetyl alcohol, a wetting agent, was employed in its pure state as a base for the cottonseed for 7 subjects (table 1). For 2 subjects, the results were negative. In 2 others, reactions developed after twelve and twenty-six hours respectively. In the remaining 3, the reactions occurred in thirty-four, forty-two and fifty-one minutes respectively. All of these reactions were weak. Slight itching and erythema were their main features, and small, poorly developed wheals occurred in 2 of the subjects.

Monkeys as Subjects: No tests were performed on monkeys with the cetyl alcohol preparation.

V. Petrolatum with 1 per Cent Potassium Carbonate.—Human Subjects: Studies were not made on human subjects with the alkaline petrolatum preparation.

Monkeys as Subjects: Tests were performed on 6 monkeys with the cottonseed-petrolatum ointment to which 1 per cent potassium carbonate had been added (table 2). Five animals reacted in from five to ten minutes, with an average of seven and one-tenth minutes for the group. One animal which failed to show evidences of absorption with the plain cottonseed-petrolatum ointment also had a negative reaction with the alkaline preparation.

COMMENT

Opinions have differed as to the comparative values of the various grease vehicles in promoting absorption of topically applied substances. For example, Moncorps [4] and Sauerland [5] reported that salicylic acid is most readily absorbed from an anhydrous wool fat base. Bliss [6] found a lard base most effective for the absorption of methyl salicylate; Brown and Scott [7] maintained that it is equally well absorbed from anhydrous wool fat, petrolatum and lard bases. Wild and Roberts [8] reported lard to be the most favorable base for the absorption of mercury.

These apparent discrepancies in the results obtained with various grease bases might be explained by variable factors, such as the concentration of the ointment, the temperature of the material or of the cutaneous site, the method of application and the site of inunction. Another factor, probably more important than any of those mentioned, is the nature of the incorporated medicament. Some substances are much more readily absorbed than others. Another prevailing impression is that animal greases, especially anhydrous wool fat, because of their similarity to the chemical composition of the fats of human skin, are most suitable for the absorption of topically applied substances.

From the results obtained in the absorption experiments on human subjects with cottonseed in petrolatum and in anhydrous wool fat (table 1), it would appear that absorption from an anhydrous wool fat base is more rapid than from petrolatum, the average absorption rate for anhydrous wool fat being nineteen and seven-tenths minutes in 17 subjects as compared with twenty-three and two-tenths minutes in 25 subjects for petrolatum. However, when the results are analyzed for the 14 subjects (table 3) on whom the inunctions were made with both bases, it will be noted that the average absorption time for both substances was about the same, i. e., seventeen and three-tenths minutes for petrolatum and seventeen and six-tenths minutes for anhydrous wool fat. In 6 of these subjects (subjects 1, 9, 14, 34, 35 and 37), the absorption was more rapid with petrolatum; in 5 (subjects 4, 10, 24, 36 and 38) the rate was more rapid with anhydrous wool fat and in 1 (subject 40), the absorption rates were the same. Two subjects (subjects 23 and 26) reacted with petrolatum and not with anhydrous wool fat. It may therefore be concluded that anhydrous wool fat and petrolatum are equally effective when serving as bases for percutaneous applications of cottonseed protein in human beings.

TABLE 3.—*Comparison in Absorption Rates in Human Subjects of Cottonseed in Petrolatum and Anhydrous Wool Fat Bases*

Subject No.	Time of Appearance of Reaction, Minutes	
	Petrolatum	Anhydrous Wool Fat
1	24	31
4	18	15
9	20	30
10	23	17
14	11	13
23	30	Negative
24	19	11
26	34	Negative
34	20	23
35	14	17
36	12	11
37	15	18
38	19	15
40	12	12

In the experiments on monkeys, 13 were tested with the petrolatum base and only 3 with the anhydrous wool fat base (table 2). The average reaction time was seven and two-tenths minutes for the anhydrous wool fat preparation as compared with nine and eight-tenths minutes for petrolatum. When the reaction times are compared for the 3 monkeys in which both bases were studied, it will be noted that not only was the average absorption time for anhydrous wool fat (seven and two-tenths minutes) shorter than

4. Moncorps, C.: Untersuchungen über die Pharmakologie und Pharmakodynamik von Salben und salbeninkorporierten Medikamenten, Arch. f. exper. Path. u. Pharmakol. **141**:50 and 87, 1929.

5. Sauerland, F.: Ueber die Resorption von Arzneimitteln aus Salben bei Anwendung verschiedener Salbengrundlagen, Biochem. Ztschr. **40**:56, 1912.

6. Bliss, A. R., cited by Harry, R. G.: Modern Cosmeticology, New York, Chemical Publishing Co., 1940, p. 53.

7. Brown, E. W., and Scott, W. O.: The Comparative Absorption of Certain Salicylate Esters by the Human Skin, J. Pharmacol. & Exper. Therap. **50**:373, 1934.

8. Wild, R. B., and Roberts, I.: Absorption of Mercurials from Ointments Applied to the Skin, Brit. M. J. **1**:1076, 1926.

for petrolatum (twelve minutes) but this precedence occurred in each instance. It may therefore be concluded that in monkeys anhydrous wool fat is a more effective base for absorption of cottonseed protein than petrolatum.

The essential oils have been shown by Macht [9] to be suitable bases for the percutaneous absorption of drugs in animals. In these studies on 2 patients, oil of anise, an essential oil, was used as an inunction base for cottonseed. In one subject (table 1, no. 34) the absorption time was equal to that obtained with anhydrous wool fat as a base but slower than that with the petrolatum preparation (oil of anise, twenty-three minutes; anhydrous wool fat, twenty-three minutes; petrolatum, twenty minutes). In the other subject (table 1, no. 36) the absorption time for oil of anise was again equal to that for anhydrous wool fat, but in this instance it was slightly shorter than for petrolatum (oil of anise, eleven minutes; anhydrous wool fat, eleven minutes; petrolatum, twelve minutes).

Oil of anise was employed as an inunction base in experiments on 4 monkeys (table 2). The average reaction time for this preparation in these 4 animals was nine and four-tenths minutes, as compared with nine and eight-tenths minutes, the average for petrolatum in 13 monkeys. In the 4 monkeys in which both petrolatum and oil of anise were tested (table 2, nos. 7, 10, 16 and 18), the average reaction time was nine and four-tenths minutes with the oil of anise as compared with ten and five-tenths minutes with the petrolatum. The oil of anise therefore appears to be more effective than petrolatum when used as a base for the inunction of cottonseed in monkeys.

In comparing the average absorption times of the oil of anise and anhydrous wool fat preparations in monkeys (table 2), it was found to be seven and two-tenths minutes for anhydrous wool fat (3 monkeys) and nine and four-tenths minutes for oil of anise (4 monkeys). In 2 monkeys (table 2, nos. 16 and 18), in which both the anhydrous wool fat and oil of anise preparations were tested at different times, the anhydrous wool fat ointment gave the more rapid absorption in each instance. Therefore, for monkeys, anhydrous wool fat is apparently a better base for the absorption of cottonseed protein than is oil of anise. In general, the results obtained suggest that oil of anise presents no advantage over petrolatum and appears to be inferior to anhydrous wool fat as an inunction base for cottonseed. However, the number of tests made is too small to permit definite conclusions to be drawn at this time.

9. Macht, D. I.: The Absorption of Drugs and Poisons Through the Skin and Mucous Membranes, J. A. M. A. **110**:409 (Feb. 5) 1938.

Many observers, including Kionka,[10] Bernstein and Herrmann,[11] Nakagawa [12] and others, have shown that alkalinization of an ointment increases the absorption of the incorporated agent. Herrmann [13] obtained similar results by alkalinizing the cutaneous site before topical application.

In the absorption experiments on 5 monkeys (table 2) in which alkalinized petrolatum was used, the average absorption time for this preparation was seven and one-tenth minutes. This was more rapid than the average obtained with the untreated petrolatum (nine and eight-tenths minutes in 13 monkeys) or with oil of anise (nine and four-tenths minutes in 4 monkeys) and as rapid as that obtained with anhydrous wool fat (seven and one-tenth minutes in 3 monkeys). In 4 animals (table 2, nos. 34, 19, 53 and 36) in which petrolatum and the alkalinized petrolatum were studied at different times, the absorption time was shorter with the alkaline ointment in every instance except 1 (table 2, no. 38). It may therefore be concluded that alkalinizing the base hastens the absorption of cottonseed protein in monkeys.

Wetting agents have recently come into use for the purpose of hastening the percutaneous absorption of medication. Duemling,[14] Herrmann, Sulzberger and Baer,[15] Mumford and Mumford [16] and others have reported favorable results with a variety of substances in vehicles containing these wetting agents.

In the experiments with cottonseed, cetyl alcohol, a wetting agent, was employed as a base for 7 human subjects. In all instances the absorption of the cottonseed was much slower than with the anhydrous wool fat base. The experiments, however, were not conclusive, since no studies were made with cetyl alcohol and other wetting agents in combination with the grease bases.

It has been recognzied for a long time that the most suitable sites for percutaneous applications

10. Kionka, H.: Die Resorption der Salicylsäure durch die Haut, Klin. Wchnschr. **10**:1570, 1931.

11. Bernstein, E. T., and Herrmann, F.: The Acidity on the Surface of the Skin, New York State J. Med. **42**:436, 1942.

12. Nakagawa, K.: On the Percutaneous Therapy, Jap. J. Dermat. & Urol. **44**:16, 1938.

13. Hermann, S.: Aufnahme und Schicksal des auf die lebende Haut applizierten Unsulins: II. Mitteilung, Arch. f. exper. Path. u. Pharmakol. **179**:529, 1935.

14. Duemling, W. W.: Wetting Agents: The New Synthetic Chemicals of Use in Finer and More Efficient Topical Dermatologic Therapy, Arch. Dermat. & Syph. **43**:264 (Feb.) 1941.

15. Herrmann, F.; Sulzberger, M. B., and Baer, R. L.: New Penetrating Vehicles and Solvents, Science **96**:451, 1942.

16. Mumford, P. B., and Mumford, R. M.: Physical Properties of Ointment Bases, Brit. J. Dermat. **53**:34, 1941.

are those that possess many fine hairs, such as the forearms, because these locations are rich in sebaceous glands. The palms and soles, because of the absence of sebaceous glands, have been considered poor sites for absorption. They do, however, possess large numbers of sweat glands, and Abramson and Gorin [17] have expressed the belief that sweat glands will permit the penetration of substances from aqueous solutions. Burgi,[18] Wild and Roberts [8] and others, however, have reported absorption of substances in grease bases from these locations.

In the experiments here reported, absorption of cottonseed protein from ointment bases occurred through the palms on many occasions. The absorption time, however, was prolonged, varying from three to four times that from other sites in the same subject. Absorption from the palms is of importance clinically, in view of the large amount of massaging and rubbing that is being done with various substances in some occupations. It is important, therefore, to keep in mind that toxic manifestations may result from the absorption through the palms or through any part of the cutaneous surface when there is continuous or prolonged contact with active substances.

17. Abramson, H. A., and Gorin, M. H.: Skin Permeability, in Cold Spring Harbor Symposia on Quantitative Biology, Cold Spring Harbor, L. I., New York, The Biological Laboratory, 1940, vol. 8, p. 272.

18. Burgi, E.: La perméabilité de la peau aux médicaments et poisons, Bruxelles-méd. **16**:1481, 1936.

SUMMARY

Petrolatum and anhydrous wool fat are about equally effective when serving as bases for the inunction of cottonseed in man. In monkeys, the absorption from the anhydrous wool fat base is more rapid than from petrolatum.

In a small series of monkeys, the percutaneous absorption of cottonseed protein from an oil of anise base was somewhat more rapid than from a petrolatum preparation but slower than from an anhydrous wool fat base.

Alkalinizing the petrolatum ointment with potassium carbonate hastened the percutaneous absorption of cottonseed protein in monkeys.

There is insufficient experimental data on cetyl alcohol as a base for the cottonseed to permit any conclusion.

The absorption through the palms of cottonseed in grease bases was demonstrated repeatedly, but this occurs at a relatively slow rate.

20 Plaza Street, Brooklyn 17.

CONTACT DERMATITIS FROM COLD PERMANENT WAVING

J. B. HOWELL, M.D.
DALLAS, TEXAS

A new method of artificially curling the hair with chemicals has recently been introduced in beauty salons throughout the country. The procedure is known as "cold permanent waving." The technic is advantageous because it seldom fails to produce waves even in women who have been unable to obtain a curl with other methods. Danger of a burn is completely removed, and the curl lasts longer, since a twist can be introduced ¾ to 1 inch (1.9 to 2.5 cm.) nearer the scalp.

Dr. Bedford Shelmire and I have recently observed in our private practice an instance of contact dermatitis from "cold permanent waving."

A middle-aged white woman received her first "cold permanent wave" during the morning of Dec. 23, 1943. That afternoon she experienced burning and stinging sensations over the sides of her neck and face and over her ears, and the next morning she noted redness of the areas. When she was in the office the following day, a mild dermatitis venenata type of eruption, consisting of slight swelling, diffuse redness, pruritus and in some areas fine vesiculation, was noted. The sites involved were the ears and the back and sides of the neck and, to a lesser degree, the face and eyelids. The dermatitis reached its maximum extent within seventy-two hours, had subsided by the end of the sixth day and has remained well to date. According to the patient's statement, she had never experienced a similar eruption. The only history of contact dermatitis was an exanthem due to poison ivy during childhood.

The cosmetologist employed the following technic in giving this patient a cold wave: Initially, a special detergent was used to cleanse the hair and scalp. Sections of hair were then moistened thoroughly with the preliminary lotion, and the hair was curled by rolling the locks on rods and fixing them in position. It required approximately twenty-five minutes to section and roll the hair of the entire scalp. After this procedure the regular cold waving compound was applied to the hair. The rolled locks of hair were brushed repeatedly until saturated with this solution. Time consumed in the step was about ten minutes. A cellophane-lined rain cap with cotton placed along the margin of the hair to absorb excess fluid was worn during the forty-five minutes the waving compound was allowed to remain on the hair. Without unrolling the curls from the rods, the operator then rinsed the hair with water and dried it by blotting with a dry towel. A third solution, termed "the neutralizer," was then applied. This was freshly prepared at the time of application and consisted of a white powder dissolved in 4 ounces (120 cc.) of 17 volume (5.1 per cent) hydrogen peroxide and diluted with tap water to make 1 quart (945 cc.). One half of the solution was applied before and the remaining portion used after the rolled curls of hair were released. The procedure required ten minutes for completion. Lastly, the scalp again received a shampoo and rinse.

Patch tests on 3 normal controls with 1 per cent concentrations of the shampoo, the preliminary lotion, the waving compound, the neutralizing solution and mixtures of these chemicals failed to evoke any reactions.

Contact tests were then applied to the forearm of the patient one month after the dermatitis of her face, neck and ears had healed. On two different occasions a positive reaction followed within twenty-four hours the application of the preliminary lotion. No reactions were observed following contact tests with the waving compound, the neutralizing liquid or the shampoo.

COMMENT

This case is an example of an eczematous contact dermatitis following within twenty-four hours a "cold permanent wave." A period of incubation of sensitivity was absent. Unquestionably, therefore, this patient was already sensitive to some ingredient in the preliminary lotion, since patch tests elicited positive reactions on two different occasions. Tests on 3 normal control subjects elicited negative reactions. Sensitization due to previous contacts with the allergen present in the preliminary solution or a closely related chemical took place without the patient's knowledge. Reexposure to this substance when she received a "cold permanent wave" precipitated the allergic response.

1719 Pacific Avenue.

CUTANEOUS LEISHMANIASIS (ORIENTAL SORE)

I. TIME REQUIRED FOR DEVELOPMENT OF IMMUNITY AFTER VACCINATION

D. A. BERBERIAN, M.D.

BEIRUT, LEBANON

Vaccination against oriental sore by the intracutaneous inoculation of living cultures of Leishmania tropica, advocated by Lawrow and Dubowskoj (1937),[1] by Berberian (1939),[2] by Senekji and Beattie (1941)[3] and by Katzenellenbogen (1942,[4] may now be considered as an accepted procedure. Persons who are vaccinated or wish to be vaccinated often ask the question, "When shall we be sufficiently immune against oriental sore to escape infection by natural routes?" To answer this question correctly, I have carried out a number of reinoculations on volunteer subjects at different stages of development of their experimental sores.

EXPERIMENTAL STUDIES

N.N.N. slope cultures of L. tropica were used for all inoculations. The site of inoculation was the anterior aspect of the thigh, about 15 cm. above the kneecap. All inoculations were strictly intracutaneous. The volume of the inoculum was always 0.2 cc. and represented approximately 500,000 leptomonad forms.

EXPERIMENT V-48.—On Oct. 31, 1938, 0.2 cc. of a suspension of living culture of L. tropica in isotonic solution of sodium chloride, representing the entire growth of a nineteen day old primary culture on an N.N.N. slope, was injected intracutaneously into the left thigh 15 cm. above the kneecap. Twenty days later a small sore, about 3 mm. in diameter, became definitely visible.

On December 13, forty-three days after the first injection of living culture, a second injection was made, 8 cm. above and lateral to the first one. The inoculum consisted of a twenty-nine day old first subculture from a twenty-five day old primary culture. Fifteen days after the second injection, it was evident that this injection also had produced a "take."

On March 16, 1938, one hundred and thirty-six days after the first injection and ninety-three days after the second injection, a third injection of living L. tropica culture suspension was made, about 8 cm. above and mesial to the first. The inoculum consisted of a suspension of a mixture of cultures of varying ages—the youngest, a fourteen day old primary culture, and the oldest, a forty-two day old second subculture—from a fifteen day old first subculture from a nineteen day old primary culture. At this time the first sore was 10 mm. and the second 8 mm. in diameter.

On June 9, 1938 three unulcerated sores were seen: The first was two hundred and twenty-one days old and was 15 mm. in diameter; the second was one hundred and seventy-eight days old and was 8 mm. in diameter, and the third was eighty-five days old and only 3 mm. in diameter. Smears from all three of the sores showed the presence of leishmania bodies.

Conclusion.—An active nonulcerated oriental sore one hundred and thirty-six days old did not protect the subject against experimental infection.

EXPERIMENT V-54.—On Nov. 12, 1938, 0.2 cc. of a suspension of living culture of L. tropica representing one half of the washing of an N.N.N. slope culture, was injected intracutaneously into the left thigh 15 mm. above the kneecap. The inoculum consisted of a twelve day old first subculture from a twenty day old primary culture.

On Feb. 15, 1939, ninety-five days after the inoculation, a small papule was visible at the site of the injection.

On March 15, one hundred and twenty-three days after the first injection, 0.2 cc. of a suspension of living culture of L. tropica was injected lateral to the first injection. The inoculum consisted of a mixture of cultures of different ages.

On September 30 the first sore was three hundred and twenty-two days old. It was about 12 mm. in diameter and had not yet ulcerated. Smears from it showed numerous leishmanias. The second sore was about 4 mm. in diameter. Leishmania bodies were present in both sores.

On December 24 the first sore was four hundred and eight days old and, as a result of a bump against a hard object, had ulcerated. The second sore did not ulcerate; it remained small, about 4 mm. in diameter.

On March 15, 1940, four hundred and eighty-nine days after the inoculation, the ulcerated sore had healed completely and had left a brownish scar; the second sore had healed without ulceration.

Conclusion.—An active oriental sore one hundred and twenty-three days old did not protect the volunteer against experimental infection. The second was smaller, did not ulcerate and healed rapidly after ulceration of the first sore.

EXPERIMENT V-49.—On Nov. 2, 1938, 0.2 cc. of a suspension of living culture of L. tropica was injected into the left thigh. The inoculum consisted of a one hundred and five day old fourth subculture from a

From the Department of Parasitology, The American University of Beirut.

1. Lawrow, A. P., and Dubowskoj, P. A.: Uber Schutzimpfungen gegen Hautleishmaniose, Arch. f. Schiffs- u. Tropen-Hyg. **41**:374-379 (April) 1937.

2. Berberian, D. A.: Vaccination and Immunity Against Oriental Sore, Tr. Roy. Soc. Trop. Med. & Hyg. **33**:87-94 (June) 1939.

3. Senekji, H. A., and Beattie, C. P.: Artificial Infection and Immunization of Man with Cultures of Leishmania Tropica, Tr. Roy. Soc. Trop. Med. & Hyg. **34**: 415-419 (May) 1941.

4. Katzenellenbogen, I.: Vaccination Against Jericho Boil, Ann. Trop. Med. & Parasitol. **36**:28-32 (June) 1942.

twelve day old third subculture from a thirty-seven day old second subculture from a seventeen day old first subculture from a four day old primary culture. Two months after the injection a definite sore about 4 mm. in diameter was visible.

On March 15, 1939, one hundred and thirty-three days after the first injection, a second injection of a suspension of living culture was given. The inoculum consisted of a mixture of cultures of different ages. At this time the first sore was about 6 mm. in diameter.

On June 18 the first sore was two hundred and twenty-seven days old. It was just beginning to ulcerate, and from it was oozing a serous fluid in which leishmanias were found. The second sore was ninety-four days old and nonulcerated.

On Oct. 10 the first sore was three hundred and forty-two days old, ulcerated and secondarily infected. It was about 15 mm. in diameter and showed no leishmanias in smears. The second sore at this date was two hundred and nine days old; it was just beginning to ulcerate, and in the smear from it occasional leishmanias could be found.

On October 25 a third injection of living culture was made. The inoculum consisted of a fifteen day old primary culture. This injection caused an immediate urticarial type of cutaneous reaction at the site of injection. The wheal was about 2 cm. in diameter and was surrounded by an erythematous zone 5 cm. in diameter. The wheal was characterized by itching; it gradually subsided within a few hours, but the inflammatory reaction persisted for several days. On the fifth day it had completely subsided, leaving behind a violaceous papule, which resembled a small oriental sore.

On November 15 the first sore was three hundred and seventy-eight days old, still ulcerated and about 20 mm. in diameter, but definitely in the healing stage. No leishmanias could be found in the smears. The second sore was two hundred and ninety-one days old; it was 15 mm. in diameter and was ulcerated; smears from it did not contain leishmanias. At the site of the third injection, given on October 25, there was a violaceous papule, slightly elevated, about 8 mm. in diameter, resembling a miniature oriental sore and showing a small central area of necrosis. Smears from it did not show leishmanias.

On December 31 the first sore was four hundred and thirty-four days old; it had just healed with a thin brownish scar. The second sore was now two hundred and ninety-one days old; it was ulcerated, but it was covered with a crust and was healing under a scab. At the site of the third injection there was a nonelevated pigmented spot about 8 mm. in diameter.

Conclusion.—Three hundred and fifty-eight days after the first injection, it was not possible to produce experimental superinfection.

EXPERIMENT V-50.—On Nov. 5, 1938, the first injection of 0.2 cc. of a suspension of a living culture of L. tropica was made into the anterior aspect of the left thigh. The inoculum consisted of an eighty day old second subculture from a ten day old first subculture from a sixteen day old primary culture.

On December 13, thirty-eight days after the first injection, there was as yet no evidence of a take. A second intracutaneous injection of a suspension of living culture was given. It consisted of a twenty-eight day old first subculture from a twenty-five day old primary culture.

On Feb. 5, 1939, three months after the first injection and fifty-four days after the second, two reddish spots were visible, one about 5 mm. in diameter and the second 4 mm. in diameter.

On May 16 the first sore was one hundred and ninety-one days old, and it was 8 mm. in diameter. Both sores were intact and showed numerous leishmanias in smears.

On July 10 the first sore was two hundred and forty-six days old, and it was just beginning to ooze. It was 12 mm. in diameter. The second sore was two hundred and nine days old and was not ulcerated. Both sores showed numerous leishmanias in smears.

On October 5 both sores were ulcerated. The first sore was three hundred and thirty-three days old; it was secondarily infected and was about 25 mm. in diameter. Smears from it did not show leishmanias. The second sore was two hundred and ninety-six days old; it was also ulcerated, and the ulcer was covered with a crust. Smears made from it did not contain leishmanias. On this date a third inoculation of 0.2 cc. of a suspension of living culture was made into the opposite thigh. The inoculum consisted of a twelve day old primary culture. This injection caused an immediate (within half an hour) reaction at the site of the injection. This consisted of an urticarial wheal about 25 mm. in diameter which was surrounded by an areola about 7 cm. in diameter. The urticarial wheal gradually faded out, but the inflammatory reaction with itching persisted for a number of days. The volunteer complained of malaise and some fever. Five days later, the inflammatory reaction had completely subsided, but at the site of the injection there was an elevated papule 7 to 10 mm. in diameter and violaceous.

On October 20, fifteen days after the third injection, the papule was still persistent but showed a whitish center. Smears from all the three lesions were free from leishmanias.

On November 5, one year after the first injection, the first sore was just healing; the second sore was still covered with a scab, and at the site of the third injection a flat pigmented spot 8 by 10 mm. was visible.

On December 20 both the first and the second sore had healed with a scar. At the site of the third injection, the pigmented spot was still visible, but it was more lightly pigmented than before.

Conclusion.—Three hundred and thirty-four days after the first injection and two hundred and forty-two days after the appearance of the sore, the subject was completely protected against artificial superinfection.

EXPERIMENT V-57.—On Nov. 12, 1938, 0.2 cc. of a suspension of living culture of L. tropica was injected intracutaneously into the anterior aspect of the left thigh about 15 cm. above the kneecap. The inoculum consisted of a twelve day old first subculture from a twenty day old primary culture.

On December 17, thirty-five days after the first injection, a second injection was given. The inoculum consisted of 0.2 cc. of suspension of a thirty day old primary culture.

On June 24, 1939 two sores were present. The first was two hundred and twenty-four days old and about 20 mm. in diameter; the second, one hundred and eighty-nine days old and about 8 mm. in diameter. Neither sore was ulcerated, and both showed leishmania bodies in smears.

On June 30, two hundred and thirty days after the first injection and one hundred and ninety-five days after the second, a third injection of 0.2 cc. of a suspension of living culture was made into the skin of the opposite thigh. The inoculum consisted of a fifteen day old first subculture from a twenty day old primary culture. This was followed by an immediate local reaction, a central urticarial wheal about 20 mm. in diameter surrounded by an areola 5 to 7 cm. in diameter. The urticarial reaction persisted only a few hours, but the

inflammatory reaction lasted for a number of days, fading completely in five days.

On July 15, fifteen days after the third injection, a small sore, 4 mm. in diameter, was visible. At this stage, the first sore, which was two hundred and forty-five days old, began to ooze a clear fluid, and the second sore, which was two hundred and ten days old, was not yet ulcerated. All three sores showed leishmania bodies in smears.

On August 10 the first sore was two hundred and seventy-one days old; it was ulcerated and crusted and was about 30 mm. in diameter. The second sore, which was two hundred and twenty-six days old, was also ulcerated, and it was about 20 mm. in diameter. The third sore was forty-one days old; it was not ulcerated and was about 6 mm. in diameter.

On October 15 the first sore was three hundred and thirty-seven, the second three hundred and two and the third one hundred and seven days old. The first sore was 30 mm. in diameter, the second 25 mm. and the third 10 mm. All three sores were ulcerated. None of them revealed leishmania bodies.

On December 15 the first sore had healed with a brownish violaceous scar; the second and third sores were covered with a scab.

On Jan. 5, 1940, all three sores were healed completely.

Conclusion.—Two nonulcerated sores, two hundred and thirty and one hundred and ninety-five days old respectively, showing leishmanias in smears, had not induced as yet sufficient immunity to prevent experimental superinfection. The appearance of a violent local cutaneous reaction at the site of the third injection and the speedy development and healing of the third sore suggest partial immunity.

COMMENT

It is evident from these 5 experiments that immunity against oriental sore is produced slowly, and it may be considered complete only when Leishmania tropica can no longer be found in the smears from the sores. This is usually the case in the ulcerated stage. It is true that a small percentage of oriental sores heal without ulceration or secondary infection; such sores, although nonulcerated, are nevertheless in the stage of regression, as leishmanias are no longer demonstrable in them. This in itself is presumptive evidence that sufficient immunity has been produced by the host to destroy all the leishmania bodies present in the sore. My results confirm and extend the observations of Senekji and Beattie (1941), who observed that in 3 subjects, 1 child and 2 adults, a natural sore developed shortly after the appearance of the artificial sore. In order to determine the immunizing value of artificial inoculation, they reinoculated 2 persons who had artificial sores in the unhealed stage, 1 who had both artificial and natural sores in the unhealed stage and 5 who had natural sores in the unhealed stage. All 8 had an artificial sore in response to inoculation.

CONCLUSIONS

Immunity against oriental sore is dependent on the appearance of the sore.

Immunity cannot be considered complete until the processes of healing are apparent. This time corresponds roughly to the time of ulceration of the sore.

It is therefore necessary to take precautions throughout the period of incubation and during the time required for the healing of the sore to protect oneself against exposure to the bites of sandflies, which are the accepted biologic vectors of the disease, and against the bites of stable flies, which may transmit the infection mechanically (Berberian, 1939).

Dr. E. W. Dennis, Chairman of the Department of Parasitology and Bacteriology, gave valuable advice and read the manuscript.

Clinical Notes

MICROSPORON AUDOUINI INFECTION OF THE EYELASHES

SEYMOUR H. SILVERS, M.D., BROOKLYN

S. N., a white boy aged 8½ years, complained of a ringworm infection of the right cheek of about two weeks' duration. When he was first examined, on Nov. 23, 1943, the infected area appeared in the center of the right cheek. It was about the size of a nickel, was red and had a raised border. The hair fluoresced under the Wood filter.

On Jan. 4, 1944 the lesion on the right cheek showed three infected hairs and was flat and only slightly red. However, it was noted during examination that the right eyelashes fluoresced under the Wood filter. A few infected eyelashes were removed and cultured at the laboratories of the Department of Health. On January 14 it was reported that the growth showed Microsporon audouini. This was reaffirmed on January 18.

Examination on January 14 showed that the lesion on the cheek had cleared and was only slightly apigmented and scaly. The right upper and lower eyelids were swollen. The conjunctiva was inflamed. Under the Wood filter, about half of the upper eyelashes and about a third of the lower eyelashes showed fluorescence. The distal ends appeared unaffected.

NOTE.—Since the report was written, the patient has been cured. Treatment consisted of manual epilation of the infected eyelashes. Examinations under the Wood filter, the first two held at weekly intervals and the last one after a month's interval, failed to show infected hair.

920 Bushwick Avenue.

From the Astoria Ringworm Clinic of the Department of Health of the City of New York.

A NEW PROTECTIVE CREAM

J. G. DOWNING, M.D.; L. M. OHMART, A.M.,* AND M. J. STOKLOSA, B.S.,* BOSTON

In industries where workers cannot avoid exposure to dermatologic irritants and where it is impracticable for them to protect their hands and forearms with gloves, protective creams have a certain amount of value in minimizing cutaneous irritations. Creams used for this purpose should contain no irritating or sensitizing ingredients; they should be capable of easy application and removal, and they should be inexpensive.

Although many protective creams are now available on the market under trade names, most of them do not meet these requirements. Since their ingredients are not specified, it is not easy to determine if irritating or sensitizing substances are present; and they should be used with caution, for they may occasionally produce a dermatitis in a susceptible person. The cost of many of these trade-marked preparations is almost prohibitive.

From a number of formulas for creams prepared by us and tested under actual conditions in industry the following has been selected as producing a preparation which most nearly fulfils the requirements of a good protective cream.

	Gm. or Cc.
Stearic acid	10.0
White wax	2.0
White petrolatum	4.5
Triethanolamine	1.5
Glycerin	8.0
Distilled water	54.0
Talc	20.0
Perfume, as desired	

Melt the white wax, the stearic acid and the white petrolatum on a water bath and heat to 85 C. Mix the triethanolamine, the glycerin and the water and heat to 85 C. Add the aqueous solution to the oily mixture, remove from the source of heat and stir until congealed. Incorporate the talc with the congealed base by levigation or milling. Perfume as desired.

For the past four months, this cream has been under trial in industries where workers are exposed to mechanical irritants and various types of grime. It has been found effective in reducing the incidence of dermatitis due to these causes. It contains no irritants or sensitizers, is easily removed, stable and relatively inexpensive and can readily be prepared in most retail pharmacies.

* From the Massachusetts College of Pharmacy, Department of Pharmacy.

GONORRHEA OF THE SKIN

H. J. TEMPLETON, M.D., OAKLAND, CALIF.

Lowry and Franks's recent report [1] of primary gonorrheal cutaneous infection caused me to recall an unusual case of gonorrhea of the umbilicus which I observed a few years ago.

The patient, a young Greek, had acute urethritis. In excreted pus gonococci were demonstrated in great numbers. Shortly after the onset of the urethritis, his umbilicus became inflamed, and a thick, creamy, purulent discharge developed. Numerous typical gram-negative intracellular diplococci were found in the pus. Long-continued treatment with mild protein silver cured the umbilical infection.

3115 Webster Street.

1. Lowry, E. C., and Franks, A. G.: Am. J. Syph., Gonor. & Ven. Dis. **27**:428 (July) 1943.

DERMATOLOGISTS OF THE UNITED STATES AND CANADA IN ACTIVE SERVICE IN THE PRESENT WAR

The following lists include the names of dermatologists who are in active service in the armed forces of the United States and Canada and in the United States Public Health Service. As such lists cannot be complete or entirely accurate at the time of publication, a supplementary list will be published later. It will include new names and changes in rank. All of the men in these lists in the United States forces are classified by the Army, Navy or Public Health Service as dermatologists.

The lists include 262 men in the Army of the United States, 70 in the United States Navy and 16 in the United States Public Health Service, a total of 348. A separate list gives the names of 7 men in the Armed forces of Canada.

The dermatologists in the United States forces were drawn from 33 states, the District of Columbia and the Territory of Hawaii. New York furnished the largest number, 80, followed by Pennsylvania with 27, California with 26, Illinois with 23, Ohio with 19, New Jersey and Texas with 17 each, Michigan with 16, Maryland with 15 and the District of Columbia with 8.

An asterisk precedes the names of men who have been certified by the American Board of Dermatology and Syphilology. Some of those who are not certified will, however, soon be eligible for examination by the board. It is requested that the editor be notified of omissions from these lists, of changes in rank or status and of any errors that may have been made.

Dermatologists Serving in the Army of the United States

Name and Rank	Home Address
Abarbanel, Milton G., First Lieutenant, M.C.	Jersey City, N. J.
Abbey, Edward A., Major, M.C.	New Haven, Conn.
Allamon, Emmett L., First Lieutenant, M.C.	Sabine Pass, Tex.
*Ambler, John V., Major, M.C.	Denver
*Anderson, Carsten R., Major, M.C.	Los Angeles
Aquilino, Armand, Captain, M.C.	Syracuse, N. Y.
Arak, Charles, First Lieutenant, M.C.	Woodside, N. Y.
Arons, Harry, First Lieutenant, M.C.	Newark, N. J.
Arthur, Robert, Captain, M.C.	Springfield, O.
Baer, Townsend W., Captain, M.C.	Pittsburgh
*Ball, Franklin I., Major, M.C. (Air Service)	Los Angeles
Barnes, Maurice C., Captain, M.C.	Waco, Tex.
*Becker, Frederic T., Captain, M.C.	Duluth, Minn.
*Benedek, Tibor, Major, M.C.	Chicago
Benson, Joseph A., Captain, M.C.	Tacoma, Wash.
*Bereston, Eugene, First Lieutenant, M.C.	Baltimore
Berg, Howard W., Major, M.C.	Albany, N. Y.
Berkowitz, Samuel, First Lieutenant, M.C.	Brooklyn
Berlind, Melvyn, Major, M.C.	Brooklyn
Berman, Dave, First Lieutenant, M.C.	Columbus, Ga.
Bloom, Manuel G., First Lieutenant, M.C.	Houston, Tex.
Boland, John P., Captain, M.C.	Chicago
Boyd, Granberry D., Captain, M.C.	Galveston, Tex.
*Brau, John G., First Lieutenant, M.C.	Abilene, Tex.
Brody, Melvin, First Lieutenant, M.C.	Canton, O.
*Brunsting, Henry A., Captain, M.C.	Toledo, O.
Burk, Joseph E., First Lieutenant, M.C.	Lecompte, La.
*Butler, Milton G., Captain, M.C.	Saginaw, Mich.
*Campbell, Henry S., Major, M.C.	Los Angeles
*Cantor, Mortimer J., Captain, M.C.	Brooklyn
Carlisle, Richard C., First Lieutenant, M.C.	Columbus, O.
Cawley, Edward P., Captain, M.C.	Jackson, Mich.
Chung-Hoon, Edwin K., Major, M.C.	Honolulu, Territory of Hawaii
Cirincione, Joseph, Captain, M.C.	Schenectady, N. Y.
Clark, George H., Lieutenant Colonel, M.C.	Oskaloosa, Ia.
*Cockerell, Earl R., Captain, M.C.	New Orleans
Cohen, Max H., Major, M.C.	Washington, D. C.
Cohen, Milton H., Captain, M.C.	York, Pa.
*Connor, William H., Captain, M.C.	Houston, Tex.
*Conrad, Adolph H., Jr., Captain, M.C.	St. Louis
Cope, Ellis P., Captain, M.C.	Little Rock, Ark.
*Cormia, Frank E., Captain, M.C.	San Francisco
*Curtis, George H., First Lieutenant, M.C.	Cleveland
Czerwinski, Anthony B., Captain, M.C.	Springfield, Ill.
*Dantzig, Louis, Major, M.C.	New York
*Davis, Marion I., Captain, M.C.	Donelson, Tenn.
*Decker, Douglass A., Major, M.C.	Allentown, Pa.
*De Oreo, Gerard A., Major, M.C.	Cleveland
Deutel, Oscar R., First Lieutenant, M.C.	Bloomfield, N. J.
Dietrich, Carlisle, Captain, M.C.	Tacoma, Wash.
Dolan, David J., Captain, M.C.	New York
Dolce, Frank A., Captain, M.C.	Buffalo
Doyle, William H., Captain, M.C.	Muskogee, Okla.
*Drake, James R., Captain, M.C.	Mountainview, Calif.
*Dwinelle, James H., Major, M.C.	Greenwich, Conn.
*Elson, Francis S., First Lieutenant, M.C.	Huntington, N. Y.
Emerson, George F., First Lieutenant, M.C.	Rochester, Minn.
*Emmett, Richard, Captain, M.C.	Brooklyn
*Epstein, Ervin, First Lieutenant, M.C.	Oakland, Calif.
Falk, Alfred B., Captain, M.C.	Chicago
Finkle, Theodore H., First Lieutenant, M.C.	New York
Flood, James M., Captain, M.C.	Philadelphia
Flood, William E., Captain, M.C.	Boston
*Foerster, Hervey A., Major, M.C.	Oklahoma City
*Foster, Paul D., Captain, M.C.	Los Angeles
*Frank, Samuel B., Major, M.C.	Cleveland
*Franks, Andrew G., Major, M.C.	New York
Freedberg, Alan P., First Lieutenant, M.C.	Salem, Mass.
Frick, John P., Lieutenant Colonel, M.C.	Kansas City, Mo.
Frumess, Gerald M., Major, M.C.	Denver
*Gandy, Daniel T., Major, M.C.	Houston, Tex.
Gewirtz, James A., First Lieutenant, M.C.	Brooklyn
*Gillespie, Emerson, Major, M.C.	Canton, O.
*Ginsberg, Julius E., Captain, M.C.	Chicago
Glassman, Herman, First Lieutenant, M.C.	Brooklyn
*Glicklich, Earl A., Major, M.C.	Cincinnati
*Goldberg, Lawrence C., First Lieutenant, M.C.	Cincinnati

Name and Rank	Home Address
*Goldman, Hyman J., Captain, M.C.	St. Louis
Gordon, James H., Major, M.C.	Silver Spring, Md.
Graham, Paul V., First Lieutenant, M.C.	Wheeling, W. Va.
*Grauer, Franklin H., Major, M.C.	New York
Green, Harry, Captain, M.C.	Tulsa, Okla.
Green, Manuel E., Captain, M.C.	Pittsburgh
Green, Ray C., Major, M.C.	San Bernardino, Calif.
*Greenberg, Samuel I., Major, M.C.	Brooklyn
Greenberg, Solomon, Captain, M.C.	Bayonne, N. J.
*Greenwald, Irving, Captain, M.C.	New Rochelle, N. Y.
Griffith, Paul R., First Lieutenant, M.C.	Berwyn, Ill.
Hahn, Joseph, First Lieutenant, M.C.	New York
Hall, James L., First Lieutenant, M.C.	Vicksburg, Miss.
Hall, Vonnie A., First Lieutenant, M.C.	Memphis, Tenn.
Harris, Hubert L., First Lieutenant, M.C.	Baltimore
*Harrison, Malcolm W., Major, M.C.	Houston, Tex.
Hawes, William J., Major, M.C.	Columbia, Mo.
*Hazel, Onis G., Major, M.C. (Air Service)	Oklahoma City
Hecht, Rudolf, Captain, M.C.	Chicago
Hegarty, Francis A., Captain, M.C.	Pittsburgh
Herman, Louis, Captain, M.C.	Ft. Custer, Mich.
*Hitschmann, Otto, Captain, M.C.	New York
Holland, Moses H., First Lieutenant, M.C.	Weehawken, N. J.
*Holman, Herbert H., Captain, M.C.	Detroit
*Holtzman, Irving N., First Lieutenant, M.C.	Brooklyn
*Hookey, John A., Captain, M.C.	Detroit
Houston, William H., Jr., First Lieutenant, M.C.	Palo Alto, Calif.
Huber, William McP., Major, M.C.	Boston
Hughes, Raymond P., First Lieutenant, M.C.	El Paso, Tex.
Illge, Alfred H., Major, M.C.	Portland, Ore.
Italiano, Anthony J., First Lieutenant, M.C.	New Orleans
Jelsomino, Samuel J., First Lieutenant, M.C.	Buffalo
Johnson, Lewis M., Major, M.C.	Lancaster, Pa.
Kahn, David, First Lieutenant, M.C.	Chicago
Kelley, William E., First Lieutenant, M.C.	Omaha
*Kelly, Don E., Captain, M.C.	Indianapolis
Kelly, Wendell C., Captain, M.C.	Detroit
Kendrick, Frank J., Captain, M.C.	Gary, Ind.
*Kennedy, Charles B., Major, M.C.	New Orleans
*Kierland, Robert R., Major, M.C.	Rochester, Minn.
Klein, Joseph B., Captain, M.C.	New York
Kline, Herman, Captain, M.C.	Atlantic City, N. J.
Knerler, Charles W., Captain, M.C.	Cumberland, Md.
Korth, Zeno N., Major, M.C.	Omaha
Kraabel, Austin B., Captain, M.C.	Seattle
*Krugh, Francis J., Captain, M.C.	Homestead, Pa.
Kwint, Joseph A., First Lieutenant, M.C.	Avenel, N. J.
*Lacksen, Frank J., Major, M.C.	Columbus, O.
*Lancaster, Augustus, Major, M.C.	Knoxville, Tenn.
*Larralde, Juan, First Lieutenant, M.C.	New York
Lawrence, Herbert, First Lieutenant, M.C.	Los Angeles
Lee, Carlton G., Captain, M.C.	Highland Falls, N. Y.
Lefholz, Rothwell, First Lieutenant, M.C.	Coral Gables, Fla.
*Lehmann, Cornelius F., Lieutenant Colonel, M.C.	San Antonio, Tex.
*Leifer, William, Captain, M.C.	New York
*Levin, Edward A., Captain, M.C.	San Francisco
Levine, Edward P., Captain, M.C.	Newark, N. J.
Lewe, Irving A., Captain, M.C.	Los Angeles
*Livingood, Clarence S., Major, M.C.	Philadelphia
Locasto, Camillo B., Captain, M.C.	Manchester, N. H.
Long, William E., First Lieutenant, M.C.	Boston
*Loomis, Elmer C., Major, M.C. (Air Service)	Dayton, O.
*Loveman, Adolph B., Major, M.C.	Louisville, Ky.
Lubowe, Irwin I., Captain, M.C.	New York
Luttinger, Daniel, Captain, M.C.	New York
Manson, Richard, Captain, M.C.	Richmond, Va.
*Mapother, Paul, First Lieutenant, M.C.	Louisville, Ky.
*Marcus, Morris D., Captain, M.C.	St. Louis
Marjey, Elmer J., Captain, M.C.	New York
Marks, Irving E., Captain, M.C.	New York
May, Murray M., First Lieutenant, M.C.	Apalachin, N. Y.
Mebel, Frederick R., First Lieutenant, M.C.	New York
Merrick, Theodore P., First Lieutenant, M.C.	Washington, D. C.
*Messina, Salvatore J., First Lieutenant, M.C.	Boston
Miller, Harry B., First Lieutenant, M.C.	Hartford, Conn.
Moore, Alfred L., First Lieutenant, M.C.	Flushing, N. Y.
*Morginson, William J., Captain, M.C.	Springfield, Ill.
Murphy, Edward L., Major, M.C.	Chicago
Murphy, Robert J., First Lieutenant, M.C.	Baltimore
Musman, David J., Captain, M.C.	Denver
McCormick, Wilbur C., Captain, M.C.	Terre Haute, Ind.
*McCuskey, John M., Captain, M.C.	Peoria, Ill.
McDonald, Frank M., Captain, M.C.	Akron, O.
McDonough, Raphael C., Captain, M.C.	Spokane, Wash.
Nelson, Lawrence M., Captain, M.C.	Minneapolis
Neumann, Adrian, Captain, M.C.	Astoria, N. Y.
*Newman, Ben A., Major, M.C.	Los Angeles
Nicholas, Leslie, First Lieutenant, M.C. (Air Service)	Philadelphia
*Nickel, Walter R., First Lieutenant, M.C.	Vancouver, Wash.
*Noun, Maurice H., Major, M.C.	Des Moines, Ia.
*O'Farrell, Norman M., Captain, M.C.	San Diego, Calif.
Olshansky, Abraham L., Lieutenant Colonel, M.C.	Albany, N. Y.
Orfuss, Abraham J., First Lieutenant, M.C.	Harrisburg, Pa.
Orsborn, Ernest V., First Lieutenant, M.C.	Chicago
Ortolano, James J., First Lieutenant, M.C.	Hoboken, N. J.
*Palmer, Robert B., Captain, M.C.	Lincoln, Neb.
*Pepple, Allen W., Captain, M.C.	Richmond, Va.
Perrin, Samuel R., First Lieutenant, M.C.	Pittsburgh
*Pillsbury, Donald M., Lieutenant Colonel	Philadelphia
*Pincus, Isadore, Captain, M.C.	New York
Pinne, George F., First Lieutenant, M.C.	Omaha
*Polsky, Morris, Captain, M.C.	Kansas City, Mo.
Porritt, Ross J., First Lieutenant, M.C.	Pontiac, Mich.
*Prunty, Francis C., Captain, M.C.	Urbana, Ill.
*Quinby, Shepard, Captain, M.C.	Buffalo
Randquist, Robert C., First Lieutenant, M.C.	Chicago
Rapp, Milton V., Captain, M.C.	Rochester, N. Y.
Ravitz, Samuel F., Captain, M.C.	Newark, N. J.
*Ray, Leon F., First Lieutenant, M.C.	Portland, Ore.
Reifler, Reuben M., First Lieutenant, M.C.	Macon, Ga.
*Rein, Charles R., Major, M.C.	New York
*Rein, Robert L., Captain, M.C.	Brooklyn
Reinard, Louis, Captain, M.C.	Los Angeles
*Reque, Paul G., Captain, M.C. (Air Service)	Durham, N. C.
*Reuter, Robert J., Captain, M.C.	Milwaukee
Richardson, Jefferson N., First Lieutenant, M.C.	Philadelphia
*Rickloff, Raymond J., Captain, M.C.	Erie, Pa.
Robbins, Jacob B., First Lieutenant, M.C.	El Paso, Tex.
Robinson, Harry M., Captain, M.C.	Chevy Chase, Md.
Robinson, Howard, Captain, M.C.	Columbus, Ga.
Robinson, Raymond C., Captain, M.C.	Baltimore
Rohrbacher, George H., Lieutenant Colonel	Stockton, Calif.
Rosenberg, Harold W., First Lieutenant, M.C.	New York
*Rosenfeld, Herbert, Captain, M.C.	New York
Rosenthal, Louis H., First Lieutenant, M.C.	Detroit
Rosenthal, Quintin, Major, M.C.	Peekskill, N. Y.
Rower, Morris L., Captain, M.C.	Utica, N. Y.
*Rubin, Albert, Captain, M.C.	Brooklyn
Rubin, Zoltan, First Lieutenant, M.C.	Astoria, N. Y.

Name and Rank	Home Address
Rubinowitz, Abraham M., First Lieutenant, M.C.	New York
Ryan, Jeremiah, Captain, M.C.	Binghamton, N. Y.
*Sachs, Harry M., First Lieutenant, M.C.	New York
*Saffron, Morris H., Major, M.C.	Passaic, N. J.
Safran, Nathaniel, First Lieutenant, M.C.	Flushing, N. Y.
Saltzman, Jacob, First Lieutenant, M.C.	Newark, O.
Samitz, Morris H., First Lieutenant, M.C.	Philadelphia
Satulsky, Emanuel M., First Lieutenant, M.C.	Elizabeth, N. J.
*Saunders, Richard L., Captain, M.C.	Buffalo
Sawicky, Herman H., Captain, M.C.	New York
Schiff, Bencel L., Captain, M.C.	Philadelphia
*Schmitt, Charles L., Major, M.C.	Pittsburgh
*Scholtz, Julius R., Captain, M.C. (Air Service)	Los Angeles
*Schonberg, Irving L., Captain, M.C.	Wichita Falls, Tex.
Schwartz, Oscar D., Major, M.C.	Detroit
*Schwartz, Walter F., Captain, M.C.	Pasadena, Calif.
*Seale, Everett R., Major, M.C. (Air Service)	Houston, Tex.
Shapiro, Abraham A., First Lieutenant, M.C.	Baltimore
*Shellow, Harold, Captain, M.C.	Chicago
Sherer, Raymond J., First Lieutenant, M.C.	New York
Showstack, Irving, First Lieutenant, M.C.	Boston
Simon, Milton, First Lieutenant, M.C.	Forest Hills, N. Y.
*Sklar, Harry, Captain, M.C.	Hempstead, N. Y.
Slaughter, John C., Jr., First Lieutenant, M.C.	Detroit
Snipes, James J., Major, M.C.	Lincoln, Neb.
*Snow, James S., First Lieutenant, M.C.	Birmingham, Ala.
*Spiller, William F., Major, M.C.	Galveston, Tex.
Stephenson, William C., Jr., Captain, M.C.	Roanoke, Va.
*Sternberg, Thomas H., Major, M.C.	Washington, D. C.
Stolar, Robert, First Lieutenant, M.C.	Washington, D. C.
Stout, Orin M., Major, M.C.	Denver
*Sullivan, Maurice, Captain, M.C.	Baltimore
*Sutton, Richard L., Jr., Captain, M.C.	Kansas City, Mo.
*Swarts, William B., Captain, M.C.	Greenwich, Conn.
Swartz, Irving, Captain, M.C.	Syracuse, N. Y.
Tobin, Louis H, First Lieutenant, M.C.	New York
Tompkins, Harold P, First Lieutenant, M.C.	Riverside, Calif.
Toomey, Thomas N., Major, M.C.	Springfield, Ill.
*Traenkle, Herbert L., Captain, M.C.	Schenectady, N. Y.
*Vogel, Harold R., Captain, M.C.	Pittsburgh
*Waisman, Morris, First Lieutenant, M.C.	Tampa, Fla.
*Walsh, Edmund N., Major, M.C.	Whiting, Ind.
*Warren, Leon H., Major, M.C.	Bethesda, Md.
Weber, Eugene P., First Lieutenant, M.C.	Olney, Ill.
*Webster, James R., Captain, M.C.	Chicago
*Weisman, Stephen L, Captain, M.C.	Paterson, N. J.
Weitz, Martin L., Major, M.C.	Jamaica, N. Y.
*Willett, Wendel M., First Lieutenant, M.C.	Des Moines, Iowa
*Wilson, Donald J., Lieutenant Colonel, M.C.	Des Moines, Iowa
Winer, Marvin N, First Lieutenant, M.C.	Buffalo
*Wolff, Max J., Major, M.C. (Air Service)	Los Angeles
*Woodburne, Arthur R., Major, M.C.	Grand Rapids, Mich
*Woolhandler, Harry W., Lieutenant Colonel, M.C.	Pittsburgh
Young, John W., Lieutenant Colonel, M.C.	Steubenville, O.
Zavoylannis, Savas J., Captain, M.C.	Brooklyn
Zippin, George J., First Lieutenant, M.C.	Albany, N. Y.

Dermatologists Serving in the Medical Corps United States Naval Reserve

Name and Rank	Home Address
*Alden, Herbert S., Lieutenant Commander (MC), U.S.N.R.	Atlanta, Ga.
*Allington, Herman V., Lieutenant (MC), U.S.N.R.	Oakland, Calif.
Anderson, Harold T., Lieutenant Commander (MC), U.S.N.R.	Buffalo
Barksdale, Edwin E., Lieutenant (MC), U.S.N.R.	Danville, Va.
Besancon, John H., Lieutenant Commander (MC), U.S.N.R.	Detroit
Boggs, Edward H., Lieutenant (jg) (MC), U.S.N.R.	New York
*Campbell, Alexander D., Lieutenant (jg) (MC), U.S.N.R.	Seattle
*Carpenter, Cedric C., Lieutenant Commander (MC), U.S.N.R.	Summit, N. J.
Cohen, Theodore M., Lieutenant (jg) (MC), U.S.N.R.	Chicago
Coomber, Ralph, Lieutenant (MC), U.S.N.R.	Chicago
Cukerbaum, Alfred R., Lieutenant (MC), U.S.N.R.	Youngstown, O.
Dement, Donald E., Lieutenant Commander (MC), U.S.N.R.	Newton, Mass.
*Derzavis, Jack L., Lieutenant (jg) (MC), U.S.N.R.	Washington, D. C.
*Director, William, Lieutenant Commander (MC), U.S.N.R.	New York
Doyle, George C., Lieutenant Commander (MC), U.S.N.R.	Duluth, Minn.
Duemling, Werner W., Lieutenant Commander (MC), U.S.N.R.	Ft. Wayne, Ind.
Fonde, George H., Jr., Lieutenant Commander (MC), U.S.N.R.	Manhasset, N. Y.
*Ford, William K., Lieutenant Commander (MC), U.S.N.R.	Rockford, Ill.
*Fox, Everett C., Lieutenant Commander (MC), U.S.N.R.	Dallas, Tex.
Fox, F. Edward, Lieutenant Commander (MC), U.S.N.R.	Fulton, N. Y.
*Frank, Louis J., Lieutenant Commander (MC), U.S.N.R.	Sioux City, Ia.
*Gilman, Robert L., Lieutenant Commander (MC), U.S.N.R.	Philadelphia
Goldstein, Marvin B., Lieutenant Commander (MC), U.S.N.R.	Youngstown, O.
*Hailey, Hugh E., Lieutenant Commander (MC), U.S.N.R.	Atlanta, Ga.
Higi, Joseph, E., Lieutenant Commander (MC), U.S.N.R.	Newark, N. J.
Hitch, Joseph M., Lieutenant Commander (MC), U.S.N.R.	Raleigh, N. C.
Hollander, Mark B., Lieutenant (MC), U.S.N.R.	Dundalk, Md.
Huber, Paul R., Commander (MC), U.S.N.R.	Glenview, Ill.
*James, Arthur P. R., Lieutenant Commander (MC), U.S.N.R.	Toledo, O.
Joyce, Stanley J., Lieutenant Commander (MC), U.S.N.R.	Detroit
Katz, Julius H., Lieutenant (jg) (MC), U.S.N.R.	Beverly, N. J.
Kaufman, Samuel R., Lieutenant Commander (MC), U.S.N.R.	Wilkes-Barre, Pa.
Kieck, Ernest G., Lieutenant Commander (MC), U.S.N.R.	Cedar Rapids, Ia.
Litterer, Ammon B., Lieutenant Commander (MC), U.S.N.R.	Miami, Fla.
Lofgren, Robert C., Lieutenant (MC), U.S.N.R.	Philadelphia

Name and Rank	Home Address
Loftis, Earl L., Lieutenant Commander (MC), U.S.N.R.	Dallas, Tex.
Lotz, John H., Lieutenant (MC), U.S.N.R.	Memphis, Tenn.
Lounsberry, R. C., Commander (MC), U.S.N.R.	San Diego, Calif.
Love, Jesshill, Lieutenant Commander (MC), U.S.N.R.	Louisville, Ky.
*Markson, Leonard S., Lieutenant (MC), U.S.N.R.	Milwaukee
Martin, James R., Lieutenant Commander (MC), U.S.N.R.	Houston, Tex.
*Miller, Thomas H., Lieutenant Commander (MC), U.S.N.R.	Detroit
*McCarthy, Lee, Commander (MC), U.S.N.R.	Washington, D. C.
*MacClatchie, Leslie K., Lieutenant Commander (MC), U.S.N.R.	Washington, D. C.
*McLaughlin, Robert R. M., Lieutenant Commander (MC), U.S.N.R.	New York
*Newton, Hiram D., Lieutenant Commander (MC), U.S.N.R.	San Diego, Calif.
Noun, Louis J., Lieutenant (jg) (MC), U.S.N.R.	Des Moines, Ia.
*Novy, Frederick G., Jr., Lieutenant Commander (MC), U.S.N.R.	Oakland, Calif.
Patterson, Charles H., Lieutenant Commander (MC), U.S.N.R.	Bedford, Va.
Peters, Erwin E., Lieutenant (jg) (MC), U.S.N.R.	Richmond Hill, N. Y.
Piaff, Richard O., Lieutenant (MC), U.S.N.R.	San Jose, Calif.
Ricchiuti, Joseph F., Lieutenant (NC) (MC), U.S.N.R.	Pottsville, Pa.
*Rubin, Lionel, Lieutenant Commander (MC), U.S.N.R.	New York
*Sandler, Isadore L., Lieutenant Commander (MC), U.S.N.R.	Washington, D. C.
*Sayer, Arthur, Lieutenant Commander (MC), U.S.N.R.	New York
Shaw, Henry C., Lieutenant Commander (MC), U.S.N.R.	Rochester, N. Y.
*Shaw, Joseph W., Lieutenant Commander (MC), U.S.N.R.	Seattle
*Shelton, Joseph M., Lieutenant (MC) U.S.N.R.	Washington, Pa.
*Silverman, Moses, Lieutenant Commander (MC), U.S.N.R.	Brooklyn
*Slepyan, Albert H., Lieutenant (jg) (MC), U.S.N.R.	Chicago
*Sompayrac, Lauren McC., Lieutenant (MC), U.S.N.R.	Jacksonville, Fla.
Stiles, Frank, Jr., Lieutenant Commander (MC), U.S.N.R.	Lansing, Mich.
*Sulzberger, Marion B., Commander (MC), U.S.N.R.	New York
*Tedder, James W., Lieutenant (MC), U.S.N.R.	New Orleans
*Tolman, Maurice M., Lieutenant Commander (MC), U.S.N.R.	Boston
Trunnell, Thomas L., Lieutenant (jg) (MC), U.S.N.R.	Waterloo, Ia.
*Twining, H. Earle, Lieutenant Commander (MC), U.S.N.R.	Philadelphia
Viecelli, James D., Lieutenant Commander (MC), U.S.N.R.	San Francisco
*Walker, Harold L., Lieutenant (MC), U.S.N.R.	Elmira, N. Y.
*Zeligman, Israel, Lieutenant (jg) (MC), U.S.N.R.	Baltimore

Dermatologists Serving in the United States Public Health Service

*Botvinick, Isadore, Passed Assistant Surgeon (R)	Detroit
*Casper, Wolfgang A., Surgeon (R)	New York
Fisher, Samuel, Surgeon (R)	Savannah, Ga.
*Gant, James Q., Jr., Surgeon (R)	Bethesda, Md.
*Goldberg, Harry C., Passed Assistant Surgeon (R)	Plainfield, N. J.
Goldsmith, Norman R., Passed Assistant Surgeon (R)	Bethesda, Md.
*Kile, Roy L., Passed Assistant Surgeon (R)	Cincinnati
*Markel, Joseph, Passed Assistant Surgeon (R)	Pittsburgh
*Pariser, Harry, Passed Assistant Surgeon (R)	Norfolk, Va.
*Peck, Samuel M., Senior Surgeon (R)	New York
Ruch, Ralph O., Surgeon (R)	Omaha
Schwartz, Louis, Medical Director	Bethesda, Md.
Shumate, Charles A., Passed Assistant Surgeon (R)	Rush Springs, Okla.
Steiger, Howard P., Assistant Surgeon (R)	Charlotte, N. C.
*Stone, Maurice J., Passed Assistant Surgeon (R)	New York
*Wile, Udo J., Medical Director (R)	Ann Arbor, Mich.

Dermatologists Serving in the Armed Forces of Canada

*Bowman, Fred B., Lieutenant Colonel, M.C.	Hamilton, Ontario
*Davidson, Andrew M., Lieutenant Colonel, M.C.	Winnipeg, Manitoba
Duncan, Allan G., Captain, M.C.	Calgary, Alberta
Mitchell, Donald S., Lieutenant Commander, M.C.	Montreal, Quebec
*Orr, Harold, Lieutenant Colonel, M.C.	Edmonton, Alberta
Williams, Donald H., Lieutenant Colonel	Vancouver, British Columbia
*Wrong, Norman McK., Captain, M.C.	

Abstracts from Current Literature

EDITED BY DR. HERBERT RATTNER

KELOIDS. A REVIEW OF THE LITERATURE AND A REPORT OF EIGHTY CASES. JOHN GARB and M. J. STONE, Am. J. Surg. **58**:315 (Dec.) 1942.

Most keloids are caused by known traumas. Syphilis and tuberculosis were not found to be etiologic factors. The exact cause is unknown, but the hormonal factor is considered important.

Treatment with roentgen rays or radium before or soon after excision of a keloid does not prevent its recurrence. Various methods, such as the injection of fibrolysin, cryotherapy (refrigeration), electrolysis and use of ultraviolet rays or grenz rays, were valueless. Among the surgical methods used but proved ineffective by the authors are scarification with the scalpel, capillary drainage by means of through and through keloidal sutures, and subepidermal excision for the purpose of causing primary union.

Roentgen rays and radium are the only effective agents for the treatment of keloids and will cure all the smaller ones except those that are radioresistant. For large keloids and those that are radioresistant, excision in the healthy tissue with the scalpel or the electrosurgical knife is preferred. The healing is promoted by aseptic technic and the use of a stimulating substance, such as ointment of scarlet red N. F. Roentgen therapy should be applied at the first sign of recurrence, but not until then, as irradiation of the involved region before and soon after excision will not prevent the formation of a keloid.

H. MILLER, Philadelphia. [ARCH. OTOLARYNG.]

THE ANTIQUITY OF CONGENITAL SYPHILIS. R. C. HOLCOMB, Bull. Hist. Med. **10**:148 (July) 1941.

The author states in a convincing way that the leprosy described by physicians of the Middle Ages is not what is known as leprosy now, but "was syphilis of the center of the face, as now seen to develop in congenital, or, as a late or 'tertiary' symptom of acquired syphilis." The triad of causation of syphilis is unique in being contagious, venereal and congenital. Modern leprosy "has never been proved by any consistently demonstrable means to be infectious, venereal or congenital, or to possess the characteristic visceral lesions seen in syphilis," but the leprosy of the Middle Ages showed the triad of symptoms just mentioned.

Bernard de Gordon's description is typical for that time, and the author presents a translation of the pertinent chapter from the Oxford manuscript of 1348. Bernard explains the hereditary origin of the disease as follows: The embryo contracts it in utero if the father was diseased or if the mother had intercourse with an infected man during the pregnancy; further, the contagion may be acquired through intercourse with a diseased woman, and, in the latent form, a woman who has had intercourse with a diseased man but does not show any symptoms may give the disease to another man.

All these features fit a diagnosis of syphilis. Furthermore, the disease was spread by prostitutes, a fact which again speaks for the diagnosis of syphilis. Several medical authors of the sixteenth century pointed out that "lues venerea" had been described long before their time. As further evidence and absolute proof of his contention, the author brings illustrations of skulls found in the cemetery of an old leprosarium on the Rue de Douai, which are preserved in the Musée Dupuytren in Paris and which have been pronounced "as surely syphilitic as it is possible for skulls to be." (Williams, H. A.: The Origin and Antiquity of Syphilis: Evidence from Diseased Bones, *Arch. Path.* **13**: 779 [May] 1932.)

Vigo's "Surgery" (1514) does not contain the usual chapter on leprosy, a chapter which is generally extensive in any medical book of that time. There is, however, a chapter (the fifth book) on morbus gallicus giving a similar description of the result of venereal infection. The author points out that the epidemics of morbus gallicus mentioned in the late fifteenth and during the sixteenth century did not exist, the reports originating in the confusion of syphilis with many other epidemic diseases; in time "the epidemic Morbus Gallicus became solely a venereal disease under the name lues venerea," and the latter soon split up into gonorrhea and syphilis.

Many sixteenth century physicians touch on such subjects as congenital syphilis and infection of nurses by syphilitic infants, their remarks being similar to those of pre-Columbian authors, though the diseases bear different names.

During the eighteenth century authorities like Jean Astruc and John Hunter disputed the existence of congenital syphilis and considerably retarded its recognition.

"For hardly 150 years has congenital syphilis received the attention . . . it deserved. The greatest progress has been made since the discovery of the Spirochete."

This article, with its clear interpretation of medieval leprosy as syphilis, is most important for the student of the history of congenital syphilis and that of syphilis in general.

ROSENTHAL, St. Paul. [AM. J. DIS. CHILD.]

THE PLASMA PROTEINS IN DISSEMINATED LUPUS ERYTHEMATOSUS. ALVIN F. COBURN and DAN H. MOORE, Bull. Johns Hopkins Hosp. **73**:196 (Sept.) 1943.

The authors note that while during life the disease process of disseminated lupus erythematosus suggests a chronic infection autopsies have failed to produce evidence for an infectious causation. These failures to detect any causative agent have suggested that the disease process is primarily a physiologic change.

Observations during life on patients suffering from this disease have revealed the constant occurrence of hyperglobulinemia involving gamma globulin. It is the reaction of this gamma globulin fraction with certain phospholipids which gives rise to false positive Wassermann and Kline reactions.

The suggestion is made that the vascular lesions and disturbances in nutrition observed in this disease may be associated with the presence of a high concentration of circulating gamma globulin. On this basis, then, disseminated lupus erythematosus appears to be a dis-

ease of disturbed cellular metabolism, perhaps associated with the presence of this reactive protein.

NELSON PAUL ANDERSON, Los Angeles.

SENSITIVITY TO TOPICAL APPLICATION OF SULFATHIAZOLE OINTMENT. ROY A. DARKE, J. A. M. A. **124**: 403 (Feb. 12) 1944.

Darke calls attention to the degree of sensitivity to sulfathiazole ointment existing among the general population.

Two hundred and eighteen patients were treated topically with 5 per cent sulfathiazole ointment. Sensitivity to it was found in 12 of them (5.5 per cent). Two patients had an exacerbation of the sensitization reaction following the oral administration of 1 Gm. of sulfathiazole.

Because this sensitivity may preclude the use of the drug in the therapy of such diseases as meningitis, pneumonia and gonorrhea, sulfonamide preparations should be used topically only when a specific need for them can be justified.

INFLAMMATORY DISORDERS OF THE SKIN OF THE FEET. JOHN F. MADDEN, J. A. M. A. **124**:743 (March 18) 1944.

There is a tendency among both laity and physicians to call all diseases of the skin of the feet fungous infections. Fewer diagnostic errors would be made if fungous infections were diagnosed as a last resort.

Contact dermatitis usually appears on the dorsa or the sides of the feet and is rarely seen on the soles. The nails and the interdigital webs are seldom involved. The usual causes of contact dermatitis are shoe dye, sock or stocking dye, leather and shoe polish.

Paronychia is usually due to pyogenic infection, fungous infection, psoriasis or syphilis. Paronychia caused by pyogenic organisms is generally secondary to ingrown toe nails.

Psoriasis may be confined to the soles or the nails for many years or a lifetime. The soles are dry and scaly and are often fissured. The eruption is more diffuse than the occupational keratoses and usually starts in childhood or later life, in contradistinction to the congenital keratoses.

Dyshidrosis is an acute or subacute inflammatory condition occurring on the hands and the feet. The lesions occur on the soles and the interdigital webs. The eruption appears as deep-seated solitary vesicles which itch intensely. The eruption frequently follows periods of overwork, nervous exhaustion or emotional disturbance and is often accompanied by hyperhidrosis.

Lichen simplex chronicus occurs on the dorsa of the feet.

The soles are frequently the sites of lesions of scabies in babies and small children. At the Ancker Hospital, benzyl benzoate has proved less satisfactory than the compound ointment of sulfur in the treatment of scabies.

Lichen planus may be seen about the ankles, the soles and the dorsa of the feet.

CUTANEOUS MANIFESTATIONS OF THE CIRCULATORY DISORDERS OF THE FOOT. RUBEN NOMLAND, J. A. M. A. **124**:747 (March 18) 1944.

The most important cutaneous manifestations that occur on the foot in association with circulatory disorders are those which occur with peripheral vascular disease. While arteriosclerosis occurs in both sexes, symptoms and signs of obliteration of arteries in the lower extremities occur almost always in men past the age of 55. Oscillometric readings give reliable information as to the status of the arterial supply.

Thromboangiitis obliterans is an obliterative arterial disease of unknown cause in which there are often superficial migratory phlebitis and vasospastic phenomena associated with the inflammatory thrombosis. It occurs at ages from 25 to 45, almost exclusively in men, and affects predominantly the Jewish race.

Acrocyanosis, chilblain circulation, pernio and erythrocyanosis crurum puellaris are probably essentially the same except for the degree of involvement. They have in common mottled cyanosis of the extremities, lowering of the surface temperature and aggravation of symptoms during cold weather. Disorders of this type are more common in women and are particularly evident in adolescent girls.

Raynaud's syndrome is a vasospastic disorder without vascular occlusion characterized by vasomotor phenomena of the extremities, superficial dry gangrene and trophic disturbances, with fairly characteristic changes in the capillary bed. Over 95 per cent of the patients are women, mostly in the age group of 17 to 35. Rather suddenly, the change being precipitated by cold or emotional disturbances, the tips of the digits become pale and exsanguinated.

Erythromelalgia is a disorder of the extremities characterized by redness, increased surface temperature and pain. An attack is provoked when the surface temperature reaches a critical level, though there are other factors, such as dependence, which also have an effect.

FUNGOUS INFECTIONS OF THE FOOT. MARCUS RAYNER CARO, J. A. M. A. **124**:751 (March 18) 1944.

The most common fungous infection of the foot is that caused by species of ringworm fungus. Trichophyton gypseum and Trichophyton purpureum are the most frequently reported causative fungi at present, while Epidermophyton inguinale and other fungi are found less often.

Dermatophytosis of the foot may present a variety of clinical pictures. Most of these may be included in the intertriginous, vesicular and hyperkeratotic groups. In most patients in whom the infection is minimal it consists of a macerated scaling between the toes, especially in the interspace between the fourth and fifth toes. When infection is produced by T. purpureum, vesicles or acute inflammation practically never occur. Usually there are sharply outlined patches involving from a small area to the entire sole. The infected skin is dull red, with fine branny scaling and no tendency toward central clearing. Itching is commonly present.

The nails may be infected by any of the ringworm fungi. The infection commonly begins under the free margin of the nail or at the sides as a grayish or dark accumulation of scale and debris which lifts the nail away from the nail bed. The overlying nail becomes opaque and yellowish and at times greatly thickened.

For treatment of mild intertriginous infection the use of antiseptic dusting powders, such as borated talc with the addition of 2 per cent salicylic acid or mild tincture of iodine, may be effective. In treatment of patients with acute vesicular eruptions great relief may be obtained by removing the tops of the vesicles with scissors and applying continuous cold wet dressings of a 1:4,000 aqueous solution of potassium permanganate or a 1:10 dilution of solution of aluminum acetate. When the acuteness of the process has subsided, stronger measures may be introduced. Fissures may be painted with a 5 per cent solution of silver nitrate. In addition, keratolytic agents,

such as diluted ointment of benzoic and salicylic acid, may be applied. Mild tincture of iodine may be used or 2 per cent solution of gentian violet ((methylrosaniline) or Castellani's paint. For the chronic hyperkeratotic form ointment of benzoic and salicylic acid to which 1 per cent thymol has been added is a time-honored remedy, and ointments of phenylmercuric nitrate (1:1500), crude coal tar (1 to 5 per cent), dihydroxyanthranol (to 1 per cent) and chrysarobin (1 to 10 per cent) are often useful. Roentgen therapy is a valuable adjunct in the treatment of dermatophytosis. Biologic therapy with trichophytin has generally proved disappointing.

The treatment of infected nails produces at best but slow improvement. In cases of infections caused by T. purpureum the prognosis is nearly hopeless. The various remedies used include ointment of benzoic and salicylic acid and Castellani's paint. The surface of the nails should be scraped before any medicament is applied. Roentgen rays should be used cautiously. Removal of infected nails on the feet is not advised, for the new nails tend to be reinfected.

Moniliasis is an infection of the skin caused by a yeastlike fungus, Monilia albicans. When the foot is involved, the lesions are often found in the spaces between the toes as shiny red moist patches of intertrigo that are sharply outlined. In treating moniliasis it is important in every case to rule out the presence of diabetes mellitus. Almost specific in the local treatment of monilial infections of the skin and nails is a 1 per cent aqueous solution of gentian violet. Roentgen rays are beneficial, especially for paronychia. Ammoniated mercury ointment (3 to 5 per cent) is often effective.

Mycetoma is the only fungous infection involving the deeper tissues that shows a predilection for the foot. Sporotrichosis, blastomycosis and chromoblastomycosis may involve the foot.

HISTAMINE-PROTEIN COMPLEXES: SYNTHESIS AND IMMUNOLOGIC INVESTIGATION: I. HISTAMINE-AZOPROTEIN. NORBERT FELL, GERTRUDE RODNEY and DONALD E. MARSHALL, J. Immunol. **47**:237 (Sept.) 1943.

The histamine theory of anaphylaxis and allergy has been the subject of a great deal of investigation during recent years. Much of the evidence obtained, particularly during the past five years, has supported the original hypothesis of Dale and Laidlaw and Lewis that histamine released from tissue cells by an antigen-antibody reaction plays a fundamental role in anaphylaxis and allergy. However, many factors of the anaphylactic syndrome and the idiosyncrasies remain unexplained, and attempts to influence these conditions of hypersensitivity by neutralizing the effects of histamine (i. e., by trying to create refractoriness with increasing doses of the compound itself or with histaminase) have given results that are not conclusive. The purpose of the present investigation was to attempt to discover whether an artificial immunity to histamine could be developed in animals and man, so that any excess histamine released by an antigen-antibody reaction would be bound by specific antibodies before it could reach the shock organs and produce the typical symptoms of anaphylaxis or allergy. This approach to the problem of the importance of histamine in hypersensitive reactions was based on the fundamental discoveries of Landsteiner regarding immunologic specificity. If histamine could be combined with a protein to form an antigenic complex in which the histamine would function as a hapten, it should be possible to develop antibodies of the desired specificity. The histamine-protein complexes described in this report were of the type used frequently by Landsteiner to obtain artificial antigens, namely, the coupling of a simple chemical compound to a protein through an azo linkage. The protein used in most instances was despeciated normal horse serum globulin prepared by the antitoxin concentration process. This protein was employed so as to eliminate as far as possible in clinical studies any risk of sensitization to common intact proteins. Histamine antigens were prepared by coupling diazotized paraaminobenzoylhistamine to various proteins. Hyperimmunization of animals with these compounds produced antibodies that were partially specific for the histamine hapten. The immunized animals displayed a decided degree of resistance to anaphylaxis.

CORNBLEET, Chicago

URTICARIA FOLLOWING A DENTAL SILVER FILLING. H. MARKOW, New York State J. Med. **43**:1648, 1943.

The author reports the case of a 41 year old nurse in whom urticaria developed immediately after an amalgam had been used in filling a tooth. The patient demonstrated a positive reaction to contact with mercury and exacerbation of symptoms on removal of fillings and on spillovers of the amalgam into the mouth. She showed complete relief after removal of all fillings containing mercury.

OCCUPATIONAL ACNE. L. SCHWARTZ and S. M. PECK, New York State J. Med. **43**:1711, 1943.

Several chemicals and occupations are listed as causative factors of occupational acne, a frequent occupational dermatitis. The largest number of workers affected are persons exposed to insoluble cutting oils. Ship building has increased this occurrence because of the use of chlorine compounds in wire insulation. The covered parts of the body are also affected because of contact with soiled work clothes.

A table of the differential diagnoses of various types of occupational acne and of acne vulgaris is given, and the histologic changes of various types of occupational acne are extensively discussed and illustrated. The mechanism of the formation of comedos and cysts is the plugging of the pores with fine particles of the chlorine compounds and the keratogenous action of the material on the lining of the gland. Then, as in acne vulgaris, foreign body granulomas occur.

General and personal measures of prevention are suggested. The treatment consists in expression of comedos, evacuation of abscesses and cysts and frequent use of skin cleansers. There is no rationale for use of roentgen rays in treatment of occupational acne.

EPITHELIOMA OF THE CHEEK DEVELOPING 15 YEARS AFTER TRICHO X-RAY TREATMENT FOR HAIR ON THE FACE. I. I. KAPLAN, New York State J. Med. **43**:1758, 1943.

Kaplan reports the case of a 46 year old woman with a squamous cell epithelioma on the left cheek, a result of roentgen ray epilation fifteen years previously.

Repeated desiccations failed to prevent recurrences. A dose of 3,500 milligram hours of radium filtered through 1.5 mm. of platinum was successful, and no recurrence was noticed one year after the treatment.

RONCHESE, Providence, R. I.

THE VARICELLIFORM ERUPTION OF KAPOSI. A. W. PEPPLE, T. W. MURRELL and R. W. FOWLKES, South. M. J. **35**:667 (July) 1942.

The authors report the cases of 3 patients (2 of them adults) with lesions which may be classified as Kaposi's varicelliform eruption.

In 1 case intracytoplasmic inclusion bodies similar to those of Guarnieri were observed in the epithelium. A keratoconjunctivitis resulted when Paul's test was done.

If the Guarnieri body is accepted as evidence of variola or vaccinia, this lends weight to the belief that Kaposi's varicelliform eruption is a manifestation of vaccinia. SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

NEWER CONCEPTS IN THE TREATMENT OF BURNS, WITH SUGGESTIONS FOR THE MANAGEMENT OF WARTIME THERMAL INJURIES. THEODORE A. FOX, U. S. Nav. M. Bull. **40**:557 (July) 1942.

In reviewing the literature since 1925 on the treatment of burns the author finds that there are many different forms of treatment, depending on the type of burn, its location, whether or not it is complicated by wounds, etc. Because of this diversity he urges that a definite standard routine treatment for burns be adopted by the United States armed forces that could be used to give the antiseptic, analgesic eschar that would provide a dam against escape of tissue fluids, stimulate normal epithelization and form a nondisfiguring scar. His review is comprehensive, and he discusses in detail the various treatments of shock associated with burns and the advantages and disadvantages of the drugs used.

He suggests that the tannic acid method, which has been in use for years, be discarded, because it can still be improved on, since tannic acid is nongermicidal and is unstable and the resulting scar is more disfiguring than that produced with either of the methods advocated. The procedures to be followed with the use of the triple dye and sulfadiazine and triethanolamine are given in detail. It is the author's impression that the sulfadiazine and triethanolamine method is more effective but more expensive than triple dye. Either of the suggested substances is germicidal, and the results obtained with their use are better than with other methods.

Triple dye consists of aniline dyes which are more effective in combination than when used singly. Its composition is crystal violet (hexamethylpararosaniline) and brilliant green (tetraethyldiaminotriphenylmethane sulfate), 1 per cent of each and 0.1 per cent solution of neutral acriflavine. Gentian violet (methylrosaniline) may be used in place of crystal violet. The sulfadiazine-triethanolamine preparation consists of 3 per cent sulfadiazine and 8 per cent triethanolamine.

Fox does not discuss gas burns. However, he feels that after decontamination they should be treated by the usual method. GELBER, Los Angeles.

PARROT'S PSEUDOPARALYSIS. R. BENOIT, Union méd. du Canada **71**:357 (April) 1942.

A 2 month old infant had not been moving his arms for a week. Movement of his arms or legs caused him pain. There was slight nonsanguineous nasal discharge. Roentgen examination revealed a destructive process in the epiphyses of the long bones of the arms and legs. Wassermann and Kahn reactions of the blood of the baby and of the parents were positive for syphilis.

Antisyphilitic treatment produced clinical and roentgenographic improvement.

DAFFINEE, Boston. [AM. J. DIS. CHILD.]

CANCER EN CUIRASSE. LOUIS SAVATARD, Brit. J. Dermat. **55**:31 (Feb.) 1943.

Cancer en cuirasse is an advanced stage of cancerous invasion of the skin (carcinoma lenticulare) of the chest wall from an underlying cancer of the breast or from recurrence in or about the scar after the breast has been removed. In the telangiectatic type the telangiectascia is first evident in the skin over the cancer or on the scar. Its progress is fairly rapid. In the nontelangiectatic type scleroderma may be the first impression of the dermatologist, and when the condition is limited to the areolar region of the nipple it may be mistaken for Paget's disease.

BLUEFARB, Chicago.

RELATION OF BLOOD ASCORBIC ACID CLEARANCE TO ARSENICAL INTOLERANCE IN SYPHILOTHERAPY. K. L. LAHIRI, Brit. J. Dermat. **55**:158 (June) 1943.

The normal ascorbic acid content of the blood of fasting subjects of both sexes has been found by the author (1943) to be 1.2 mg. per hundred cubic centimeters, of normal women 0.72 mg. and of women before and after parturition (Lahiri and Rud, 1943) 0.9 mg. and 0.52 mg. respectively, all the subjects being Indians.

This investigation was undertaken to find out the relation of blood vitamin C clearance to arsenical intolerance. Five hundred milligrams of ascorbic acid in solution was given intravenously and the arsphenamine solution injected within fifteen minutes. In normal subjects the maximal vitamin C level of the blood at fifteen minutes was 4.5 mg. per hundred cubic centimeters, and in arsenical-intolerant syphilitic subjects, 2 mg. (This was estimated by drawing 2.5 cc. of venous blood and mixing with 5 cc. of distilled water plus 2.5 cc. of 20 per cent trichloroacetic acid and filtering. The filtrate was titrated against 2, 6-dichlorophenolindophenol at about 0 C.)

No signs and symptoms of arsenical intolerance recurred in a group of 32 syphilitic subjects having an average blood level of vitamin C of 0.21 mg. per hundred cubic centimeters.

HONEYCOMB ATROPHY. LOUIS SAVATARD, Brit. J. Dermat. **55**:259 (Nov.) 1943.

The author describes a case of "honeycomb atrophy" in a girl aged 18. In the United States this disease is known as folliculitis ulerythematosa reticulata. The outstanding characteristic feature is the symmetric honeycomb atrophy of the skin of the cheeks, but this atrophy may not be limited to the cheeks and may extend to the ears and forehead. Erythema may be present or absent. Telangiectasia is also variable. Comedos and horny follicular plugs, usually present though not necessarily so, may disappear later. Papules, pustules and seborrhea are absent. Histologically there is hypoplasia of the sebaceous glands, and the picture suggests a follicular dystrophy of congenital origin. The author treated his patient with two applications of thorium X with a three months' interval between them, with apparently good results.

THE TREATMENT OF PLANTAR WARTS BY FORMALIN. SYDNEY THOMSON, Brit. J. Dermat. **55**:267 (Nov.) 1943.

The author treated 39 patients with plantar warts with a solution of formaldehyde. Roentgen rays or radium had been used unsuccessfully for 8 of them. Of the whole series only 6 patients were lost to sight. Of the remainder, treatment failed for only 2 girls, in their late teens. The technic was as follows: A 3 per cent concentration of solution of formaldehyde B. P. was used, some being poured into a doll's saucer or doll's plate, in order that the heel or anterior part of the foot could rest in the solution without wetting the thinner skin on the top of the foot. The patients were instructed to soak the area for ten minutes each night in this way. No other lotions, ointments, plaster or medicaments were used. In most cases the pain disappeared after seven to ten days, while at the end of three weeks the warts showed as white macerated plugs embedded in the epithelium. These could easily be scraped away by means of a forceps, soft epithelium being left in the depths of the crater. While in some cases the areas were clear and apparently cured within three to four weeks, in a few the changes did not occur until seven or eight weeks had passed.

A NOTE ON A CASE OF BLASTOMYCOSIS CURED BY SULPHAPYRIDINE AND SULPHATHIAZOLE. M. ALBERT, Brit. J. Dermat. **55**:294 (Dec.) 1943.

The author reports a case of blastomycosis in a man aged 50 who was cured by sulfapyridine and sulfathiazole. Treatment consisted of administration of 80 Gm. of sulfapyridine in sixteen days followed by a rest period of fourteen days. Sulfathiazole was then given, 70 Gm. in sixteen days, followed by another rest period of fourteen days. Sulfathiazole, 36 Gm. in eight days, was given in the third course. Locally, 25 per cent solution of sodium sulfate was used as a dressing. Remarkably rapid progress was made during the first two courses, by the end of which most of the lesions had disappeared. During the rest periods there was no evidence of healing; that is, the local application of sodium sulfate was not in itself effective.

BLUEFARB, Chicago.

BELL'S PALSY AND HERPES ZOSTER. JOHN D. SPILLANE, Brit. M. J. **1**:236 (Feb. 15) 1941.

Spillane presents an interesting report on a group of cases of herpes zoster involving various segments, thoracic and lumbar. In 1 case involvement of the trigeminal nerve was associated with ipsilateral facial paralysis. The eruption was confined to the second and third divisions of the trigeminal nerve, but auricular zoster did not develop. The explanation is that the herpetic virus had involved both the gasserian and the geniculate ganglion and that the involvement of the former produced the eruption and that of the latter the facial paralysis. The order of involvement is not definitely known. There was also loss of taste of the same side of the tongue, and this is explained by an extension of the infection to the cell stations in the geniculate ganglion of the fibers of the chorda tympani nerve. The absence of auricular herpes is interesting because it indicates that herpetic invasion of the geniculate ganglion can take place without cutaneous manifestation in the distribution of its sensory fibers and points to the conclusion that facial palsy may be the only sign. This points to the interesting conclusion that in some cases Bell's palsy may be due to herpetic infection of the geniculate ganglion. This is further borne out by 1 case in which Bell's palsy occurred in a man who was in contact with a group of persons of whom several had herpes.

APPEL, Lynn, Mass.

ACTINOMYCOSIS. CHARLES PICKNEY, Proc. Roy. Soc. Med. **25**:686 (Aug.) 1942.

Pickney reports a case of actinomycosis in a boy aged 12 years.

WILLIAMSON, New Orleans. [AM. J. DIS. CHILD.]

HEMORRHAGIC PURPURA DUE TO TOXIC EFFECTS OF QUININE SALTS. R. P. VARAS, Bol. Soc. cubana de pediat. **14**:49 (Feb.) 1942.

The author reports a case of hemorrhagic purpura in a white child aged 4 years. The child had been treated with quinine salts for a febrile condition on the suspicion that the elevation of temperature was due to a malarial condition. Shortly after the use of this remedy hemorrhagic manifestations in the form of purpuric spots appeared on the inferior extremities and extended to the trunk and face. There was also gingival bleeding.

The author ascribes the appearance of purpuric manifestations to the toxic effect of the quinine salts. The use of liver extract, vitamin K and calcium chloride by mouth, with suitable dietary measures, completely restored the child within a short time.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

Society Transactions

CLEVELAND DERMATOLOGICAL SOCIETY

E. W. NETHERTON, M.D., *President*

J. E. RAUSCHKOLB, M.D., *Secretary*

J. R. DRIVER, M.D., *Reporter*

March 25, 1943

Sarcoidosis. Presented by DR. CLYDE L. CUMMER, DR. CHARLES G. LAROCCO and (by invitation) DR. E. S. RAMBOUSEK.

H. W., a Negro aged 47, presents a nodular almost translucent ham-colored growth around the right naris of nine months' duration. More recently the palmar surfaces of two fingers of the right hand have shown almond-sized dusky red indurated lesions. Many of the superficial lymph nodes have shown moderate enlargement.

He was admitted to St. Vincent Charity Hospital on Feb. 2, 1943 for observation and remained for two weeks. During this period his temperature was within normal limits.

The results of a urinalysis and the hemogram were normal. Determinations of the blood chemistry showed urea nitrogen, creatinine and calcium to be present in normal amounts. The values for blood chlorides on two occasions were 647 and 561 mg. per hundred cubic centimeters, respectively. The total proteins were increased in amount, and the ratio was reversed. The Wassermann and Kline reactions of the blood were negative. The intracutaneous tuberculin test elicited a negative reaction with a 1 : 10,000 dilution, and only a small papule was produced with a dilution of 1 : 1,000.

Fluoroscopic and roentgenographic examination of the intrathoracic organs on Sept. 18, 1942 showed rather massive bilateral enlargement of the hilar and paratracheal lymph nodes. The lungs showed no evidence of parenchymatous pulmonary disease. The examination indicated that the mediastinal lymph nodes are also the sites of lesions of Boeck's sarcoid. Osseous changes were looked for in the terminal phalanges, but none were found.

On Feb. 4, 1943 roentgenologic examination of the lungs showed no abnormalities. Examination of the left hand and fingers showed no evidence of osseous lesions. The right foot and toes showed a somewhat cysticlike expansion of the head of the proximal phalanx of the small toe, some slight fragmentation of the terminal phalanx of the fourth toe and some spotty zones of rarefaction in the second and the great toe, changes interpreted as being associated with Boeck's sarcoid.

Histologic examination of tissue from the tumor on the nose showed a circumscribed subepithelial accumulation of fibrous tissue, fibroblasts and lymphocytes. Epithelioid cells and giant cells were lacking. The appearance of the lesion was compatible with but not diagnostic of sarcoidosis. Histologic examination of an epitrochlear lymph node showed numerous tuberculoid lesions composed of fibroblasts and giant cells for the most part. There was absence of caseation necrosis. The appearance conformed with a diagnosis of Boeck's sarcoid.

DISCUSSION

DR. H. J. PARKHURST, Toledo, Ohio: I feel that this patient clearly shows the picture of sarcoidosis, including the negative reaction to tuberculin.

DR. E. W. NETHERTON: The roentgenogram of the chest showed the changes in the hilus of each lung which are characteristically seen in sarcoidosis.

DR. E. S. RAMBOUSEK (by invitation): I think the interesting feature of the case is that the laboratory findings are consistent with the diagnosis. The laboratory diagnosis of sarcoidosis was developed by Harrel Longcope and others, and they showed that if there is anything characteristic of the disease it is the elevation of the total protein with reversal of the albumin : globulin ratio. This case shows that. We performed 2 biopsies—one of the nasal lesion and one of an epitrochlear node. The node was particularly characteristic, as has been described on numerous occasions. The nodes were typical of Boeck's sarcoid.

Sickle Cell Anemia. Presented by DR. CLYDE L. CUMMER and DR. CHARLES G. LAROCCO.

I. O., a Negro aged 35, presented from the department of dermatology and syphilology, St. Vincent Charity Hospital, since December 1940 has had itching and swelling of the right leg. These symptoms had been preceded for several years by shortness of breath and palpitation. Ulcers developed on both legs in July 1942. Coin-sized circular atrophic scars on the lower portions of both legs have resulted.

On physical examination, a blowing cystolic cardiac murmur is heard over the apex. The scleras are icteric.

Sickle cells have been found in the peripheral blood on three occasions. A recent hemogram showed 2,000,000 erythrocytes and 9,200 white blood cells per cubic millimeter. The hemoglobin content was 7.9 Gm. per hundred cubic centimeters. The differential count showed 62 per cent polymorphonuclear neutrophils, 33 per cent lymphocytes and 5 per cent monocytes.

Blood smears showing sickling of the erythrocytes were demonstrated.

DISCUSSION

DR. C. L. CUMMER: I think that Dr. Parkhurst called attention to a small soft nodule on the shin which the patient states appeared yesterday or today. If this is the area of infiltration which possibly precedes the ulcer, it will be something to study and follow, for I have not been able to find anywhere a description of an early lesion before it breaks down.

This patient was not presented for dermatologic reasons but because we thought the members might be interested in the blood picture. The fresh blood film was taken yesterday and kept until today. It was rimmed with petrolatum and will last two or three weeks prepared that way. When blood is first taken, one does not always find as many of the sickle cells as expected. Today you probably noticed some that were almost stellate. Also a film of blood taken yesterday was stained with Wright's stain, and is presented to show what the stained films looked like.

DR. C. G. LAROCCO: The age of this patient is of interest. Thirty-five years is near the upper age limit for a person with sickle cell anemia. This man looks rugged and healthy, and he may have many more years ahead of him.

DR. E. W. NETHERTON: Patients with this disease often have infarction of the spleen. Has there been any acute episode that would indicate that this complication had occurred in this case?

DR. C. L. CUMMER: The patient has had symptoms attributed to his heart, and he has an apical cardiac murmur.

DR. E. W. NETHERTON: Sickle cell anemia is usually encountered in Negroes.

DR. C. L. CUMMER: There are on record only a few cases in which the patient was not a Negro but was of Mediterranean stock.

DR. E. W. NETHERTON: Certainly any Negro that has scars on the legs like this should be studied for sickle cell anemia.

Neurofibromatosis (Three Cases Presented from the Department of Dermatology and Syphilology, St. Vincent Charity Hospital). Presented by DR. CLYDE L. CUMMER and DR. C. G. LAROCCO.

CASE 1.—J. H. a Negro aged 20, complains of "lumps on the body" which have been present for at least seven or eight years. He has had the usual diseases of childhood. His father is, apparently, similarly afflicted but refuses to be examined.

The patient's face is irregularly discolored and shows a papular dermatitis, the result of exposure to dust and oil in a foundry. On the trunk are numerous café-au-lait spots and hard firm pea-sized to hickory nut–sized tumors. A curious cordlike arrangement of tumors may be seen along the course of the left ulnar nerve. They appear to be attached to the nerve sheath.

Histologic examination of a tumor showed it to be composed of vascular and fibrous tissue. The fibrous tissue was loosely arranged, the fibrils interweaving without the formation of any definite pattern. The individual cells varied somewhat; some were slightly thickened, and others were slender and curving in outline. The tissue was circumscribed by a fibrous capsular portion. Adipose tissue and nerve tissue were not seen. With Van Gieson's stain the fibers appeared pink. The blood vessels and blood cells assumed a yellow color. The appearance of the tumor was compatible with the diagnosis of neurofibromatosis, the tumor in this case being essentially of the fibromatous type.

CASE 2.—A. M., a Negro aged 17, has had tumors on his body for many years. He has had the usual diseases of childhood.

Various-sized tumors are irregularly disseminated over the cutaneous surface. Most of them are located on the upper extremities; a few are present on the trunk, face and neck. The lesions are pea sized to walnut sized, except for an unusually large flabby tumor occupying the ulnar side of the right palm. The cutaneous surface is covered with pinpoint spots of pigmentation and occasional larger pigmented lesions.

CASE 3.—M. P., a white woman aged 54, complains of bumps on her body of twenty years' duration. Her mother had a clear skin, and she knows nothing concerning her father. One sister and three brothers are not similarly affected.

Innumerable tumors ranging in size from that of a pea to that of a hazelnut cover most of the body. Many are pedunculated. There are various-sized café-au-lait spots on the trunk.

DISCUSSION OF THE PRECEDING THREE CASES

DR. HUGO HECHT: There is no doubt that cases 2 and 3 are instances of von Recklinghausen's disease. In regard to case 1, not only the location but the whole appearance is different and I believe it is not the same disease. Most impressive are the painful nodes. I think it resembles lipomatosis dolorosa. In this case the biopsy does not show the typical picture of neurofibromatosis. From the clinical viewpoint, neurofibromas are soft and when touched give the feeling of a pillow. In von Recklinghausen's disease there is no resistance when a lesion is touched by the finger, but in this case it is different. The nodes are hard and painful when touched, and this is typical of Dercum's disease.

DR. H. J. PARKHURST, Toledo, Ohio: I felt at first that the first patient did not have von Recklinghausen's disease, but I have changed my mind because he presents café-au-lait spots identical with those of the second patient and the latter, in addition to the typical soft nodules of von Recklinghausen's disease, also presents several firm tender nodules similar to those of the first patient. Therefore I now consider that both cases are instances of von Recklinghausen's disease, but the first is not of the typical variety.

DR. J. R. DRIVER: I agree with Dr. Parkhurst. I was impressed with the similarity of the subcutaneous nodes in case 1 with the ones in case 2. The second patient had a linear subcutaneous nodule in the groin that was extremely tender on pressure. The first patient also had linear nodules, and many of them were extremely tender on pressure. I believe the tenderness of these subcutaneous neurofibromas is due to the nerve tissue present. The second patient had a baggy tumor on the side of the hand sometimes spoken of as a *ranken neurom*. This type of lesion is frequently seen in cases of neurofibromatosis.

DR. R. E. BARNEY: Dr. LaRocco has apparently exerted every kind of pressure to obtain a second biopsy in case 1. Assuming that both of the patients have von Recklinghausen's disease, it is interesting to note the large number of pigmented macules with relatively few nodules in the one case while in the other the reverse is true. I wonder if the pain in association with the nodules in case 1 is entirely real. It seemed that the pain on firm pressure was not nearly so great when the patient's attention was diverted.

DR. C. L. CUMMER: In answer to Dr. Barney's suggestion, I feel that the patient's protestations of tenderness and pain are genuine, because he had the same reactions when we palpated the nodules in the clinic in the absence of any one to impress. I believe there is a genuine tenderness over the lesions.

Mycosis Fungoides. Presented by DR. E. W. NETHERTON.

Mrs. A. W., a Negress aged 32, states that nine years ago pruritic scaly areas developed on her shoulders. Similar lesions continued to develop until most regions of the cutaneous surface were involved. For eight years the lesions remained unchanged. Itching was troublesome, and treatment with many topical remedies and internal medication failed to influence the course of the eruption.

During the summer of 1942 the scaly plaques began to enlarge and become thickened, and nodules developed on the face, neck, trunk and arms. The nodules have continued to develop, and the surfaces of some of the large tumor masses have become moist as a result of superficial ulceration.

Physical examination shows enlargement of inguinal lymph nodes. The liver and spleen are not palpable. The mucous membranes of the mouth and throat are normal.

There is a polymorphous eruption involving most of the body, and only a few small areas of normal skin remain. The face is almost entirely involved by a nodular infiltrative process. There are round and gyrate well demarcated smooth infiltrated plaques on the arms and trunk. Cutaneosubcutaneous doughy nodules varying from the size of a lima bean to that of a hazelnut are disseminated over the trunk and upper extremities. Most of the skin not involved by the nodules and infiltrated plaques is covered with large oily, waxy, loosely adherent scales. The lower extremities are involved by this exfoliative process. There is edema of the lower portions of the legs. There are no bullae or vesicles.

The hemogram showed 4,560,000 erythrocytes and 12,350 white blood cells per cubic millimeter. The differential count showed 55 per cent neutrophils, 26 per cent eosinophils, 1 per cent basophils, 12 per cent lymphocytes and 6 per cent monocytes. The Wassermann and Kahn reactions of the blood were negative. The urinalysis showed normal conditions.

Histologic examination of an infiltrated plaque from the left supraclavicular region showed that the epidermis was atrophic owing to the pressure of the infiltration in the upper part of the corium. The epithelium over the dense part of the infiltrate consisted of three or four cells. The basal layer was intact. The interpapillary pegs were obliterated. There was a dense cellular infiltration involving the papillary and subpapillary layers and the upper two thirds of the corium. The infiltrate had a loose connective tissue reticulum. The cellular infiltrate was polymorphous. It consisted of fibroblasts, epithelioid cells, lymphocytes and occasional plasma cells and a few polymorphonuclear leukocytes and eosinophils. There were large round or oval-shaped cells with pale-staining nuclei throughout the infiltration. Many cells were pyknotic. Giant cells were not observed. Sebaceous glands were not seen. However, sweat glands were present and normal. There were a few mitoses, and chromatophores were present in the papillary layer.

DISCUSSION

DR. R. E. BARNEY: What is the longest time that mycosis fungoides has been known to exist? I believe that Dr. H. L. Keim reported the case of a patient who had the disease for eighteen years. The duration of the tumor stage is relatively short.

DR. E. W. NETHERTON: Ormsby stated that the premycotic stage may last months or years. The infiltrative stage runs a more rapid course. This woman says that she had scaly pruritic lesions for eight years and that in only the last year did the infiltrative lesions which are present today develop.

DR. C. G. LAROCCO: The interesting feature to me in this case is the race of the patient. In all the years that we have been observing Negro skins at St. Vincent Charity Hospital, I cannot recall a single case of mycosis fungoides in a Negro. I agree with the diagnosis.

DR. E. W. NETHERTON: The disease also is more common in men than in women.

DR. H. G. MISKJIAN: How common is it in the Negro race?

DR. E. W. NETHERTON: It is extremely uncommon. Ormsby's textbook says that it is more common in men than in women and is less common in Negroes than in white persons. I believe this patient is a full-blooded Negress.

DR. C. L. CUMMER: I have never seen a case here in the hospital in the sixteen years I have been here, and we see a rather large number of Negroes with diseases of the skin.

Poikiloderma of Civatte. Presented by DR. E. W. NETHERTON.

Mrs. W. N., aged 48, states that she had never had an eruption on her face prior to a year ago. She dates her trouble from an intense tanning of the skin due to exposure to sunshine while she was on a trip to Florida. On her return home the tan disappeared, but there was left an unusual hyperpigmentation of the face, for which she is presented.

The present eruption was not preceded by an acute dermatitis of the face. She has always used a good grade of cold cream and of face powder and has been in the habit of applying these preparations to the face each morning.

Examination shows a finely reticulated brownish to violaceous pigmentation involving all of the forehead and face. The pigmentation fades out gradually as it extends onto the lateral and anterior surfaces of the neck. Small dilated vessels are present on the cheeks. There seems to be slight atrophy of the skin on the sides of the face. There is no scaling, and there are no signs of an inflammatory infiltration in the skin. A general physical examination revealed nothing worthy of note, and the hemogram was normal.

DISCUSSION

DR. J. R. DRIVER: I think it might be helpful for Dr. Netherton to get an analysis of the face powder and cream, in order to see if there is any bismuth or mercury in them. These substances are a causative factor in some cases.

Keratosis Palmaris et Plantaris. Presented by DR. CLYDE L. CUMMER.

J. M., a white man aged 26, states that his eruption affecting the palms and soles began in December 1941 or slightly earlier. It is attributed by him to the handling of irritating materials in his work as a plater. There is no history indicative of a similar eruption in other members of the family. However, the patient is an only child, and both parents have died.

The palms are the sites of a severe hyperkeratosis having a yellow tinge. The thickening of the skin is especially noticeable in the crotch of the thumbs, on the "heel" of the hands and over the distal ends of the metacarpal bones. The color change stops sharply at the base of the hand. The finger nails are greatly thickened and yellow and in the distal portions seem to be detached from the nail beds. The soles and the toe nails are similarly affected.

The Kline exclusion test of the blood elicited a negative reaction. Suitably treated material from one great toe nail and from one finger failed to show mycelium or spores.

An ointment containing 3 per cent salicylic and 6 per cent benzoic acids has been employed, with some benefit

DISCUSSION

DR. E. C. STERN: I think the disease is dermatomycosis. The patient is too young for a keratosis. He has a thick horny layer under his nails. Were scrapings taken from there? If one scrapes rather deep under the nails, fungi may be found.

DR. C. L. CUMMER: I think the involvement of the nails is too universal to explain as a dermatomycosis. I find in Pardo-Castello's monograph that just such changes as these occur in keratosis palmaris et plantaris. The only thing that raised a question in my mind was the late appearance of the eruption. However, I think it can begin later in life as well as in infancy.

DR. HUGO HECHT: This case is an instance of keratosis palmaris et plantaris hereditaria. I, myself, am from a family in which it occurs, and I was unaware of it until I was 35 years of age. At that time I heard that the older dermatologists in Prague were familiar with this disease and called it by my mother's family name—"Piesen's Disease." I traced my mother's family tree for six generations, which included about 250 members. I presented it at the Congress of the Deutsche dermatologische Gesellschaft in 1929 at Koenigsberg, Germany. The disposition to the disease is hereditary, and occasionally the eruption is initiated by an external irritation. It started in my oldest son at the age of 10 with an irritation caused by a ring he was wearing on the fourth finger, and from here it spread over the whole palm in spite of treatment.

The patient presented today does not know anything about his family because his parents died when he was young, but I think his eruption also is hereditary.

DR. E. C. STERN: Were there changes in the finger nails and toe nails?

DR. HUGO HECHT: He could have keratosis and onychomycosis.

DR. C. B. NORRIS, Youngstown, Ohio: The patient says that the eruption began a year and a half ago. Dr. Hecht spoke of irritation being a causative factor. It may be, since he works as a plater, that cadmium is the irritating substance.

DR. E. W. NETHERTON: The absolute symmetry of the eruption would be against a fungous infection. There is also a sharp line of demarcation along the sides of the hands, which is characteristic of keratosis palmaris et plantaris. I think that the diagnosis in this case is as presented, and if there is a fungous infection I believe that it is coincidental.

Lichen Sclerosus et Atrophicus. Presented by DR. CLYDE L. CUMMER.

J. S. W., a white woman aged 28, has had an eruption for five years. The lesions, 5 mm. or less in diameter, are situated on the upper posterior portion of the trunk, where they are grouped irregularly. When first seen, in July 1942, they were flat, glistening, white and depressed and many were confluent. Some appeared as atrophic scars. On the upper inner quadrant of the right breast is a similar lesion, 3 by 4 cm. in size, with an elevated border and the appearance of cordlike bands running under the surface.

On the peroneal aspect of the right leg is a coin-sized flat and slightly depressed scar, said to have resulted from similar lesions.

The Kline reaction of the blood was negative.

Histologic examination showed slight hyperkeratosis, flattening of the dermal papillae, localized edema of the upper layers of the dermis and a mild nonspecific chronic inflammatory reaction in the dermis. The lower layers of the dermis showed no edema, degeneration or inflammatory infiltration.

A bismuth preparation has been given by intramuscular injection. There has been decided improvement as seen in flattening of lesions and lessening of scaling. Many of the lesions now have the appearance of atrophic scars.

DISCUSSION

DR. H. J. PARKHURST, Toledo, Ohio: I thought this was a case of lichen sclerosus et atrophicus, and according to my conception it is not related to lichen planus. In this case the lesions appear in the favorite location of this disease. Most of those that I have seen have been on top of the shoulders.

DR. E. W. NETHERTON: The biopsy is important in a case like this. The biopsy slide presented today shows the changes pointed out by Dr. Montgomery that occur in lichen sclerosus et atrophicus. There is a zone of homogeneous pale-staining degeneration in the papillary and subpapillary layers of the corium. This change rules out the diagnosis of lichen planus with atrophy.

MINNESOTA DERMATOLOGICAL SOCIETY

S. E. SWEITZER, M.D., *President*

H. A. CUMMING, M.D., *Secretary*

Minneapolis, Oct. 8, 1943

Lupus Erythematosus Involving Eyelids and Conjunctiva. Presented by DR. S. EPSTEIN, Marshfield, Wis.

The patient, a woman 32 years of age, was first seen on June 8, 1943. She gave the following history: For about six months round lesions had appeared on her scalp, behind her ears, on her nose, on her chest and on her back. The lesions grew larger and more conspicuous as time passed. About a year ago she had a sty on her left lower eyelid; it was incised, but the wound never healed completely. During the past few months, apparently simultaneously with the other eruption, redness and inflammation appeared on the lower lid. The lesion increased in size, and the eye became sore. About the same time the patient noticed an eruption of the buccal mucous membranes.

At the time of the examination, the patient presented several more or less round lesions in the aforementioned areas. They consisted of decidedly infiltrated reddish plaques, some of which had a central hyperkeratosis. Involution has occurred in a number of these lesions following a course of injections of a bismuth preparation. Two new ones have appeared around the left eye, one at the site where the glasses would irritate the skin.

The left eye presented the picture of a conjunctivitis and a peculiar dermatitis of the lower lid. There was a somewhat hard edema with infiltration. The center was rather atrophic and white. The swelling of the lid has diminished, but otherwise the lesion has not changed materially.

The mucous membrane of the mouth showed symmetric confluent lesions composed of white plaques. There were some infiltration and extensive erosion at the center. These lesions have improved considerably.

The patient is in good general health. The blood count was normal, and a focus of infection has not been found. The histologic examination of a section of a lesion from the back was compatible with the diagnosis of lupus erythematosus. Lichen planus could not be ruled out definitely.

DISCUSSION

DR. S. EPSTEIN, Marshfield, Wis.: This case seemed interesting on account of the involvement of the eyelid, which is of rare occurrence. I cannot remember having seen involvement of the conjunctiva before. A few weeks after I saw this patient, I had another one with a typical lupus erythematosus and a patch on the right lower eyelid extending inside to the conjunctiva of the lid. I shall appreciate therapeutic suggestions for the stubborn lesion of the eyelid.

DR. S. E. SWEITZER, Minneapolis: The cautery might be used for the lesion in the mouth.

DR. H. MONTGOMERY, Rochester: You might try solid carbon dioxide for the eyelid.

Epithelioma of the Anus. Presented by DR. S. E. SWEITZER, Minneapolis.

A. S., a white woman aged 65, was first seen in consultation by the dermatologic staff of Minneapolis General Hospital on Oct. 6, 1943, when she complained of an ulcer of the perianal region which had been present for six months. There was little pain and only a little bleeding.

The Wassermann reaction was negative. A roentgenogram of the lungs revealed nothing abnormal except an old fracture of the seventh rib on the right side, which was interpreted as a possible metastasis.

Examination reveals a well developed and well nourished woman with an ulcer measuring 5 by 7 cm. in the perianal region. The floor of the ulcer is "velvetlike" granulation tissue, and the periphery is rolled and indurated. There is involvement of the posterior wall of the vagina.

DISCUSSION

DR. S. E. SWEITZER, Minneapolis: It is hard to understand why any one would neglect anything of such a serious nature for so long, especially something which obviously must have caused a great deal of discomfort.

DR. H. E. MICHELSON, Minneapolis: It has always amazed me that in spite of the fact that one sees many patients with pruritus ani one rarely sees an epithelioma of the anal region; but when such an epithelioma is seen, it is difficult to handle and highly malignant.

DR. H. MONTGOMERY, Rochester: The histologic examination revealed a squamous cell epithelioma of grade 2. Usually when the anus is involved, the process is highly malignant, with a histologic grade of 3 or 4, and the tumor usually extends onto the mucous membrane and lower portion of the bowel. The prognosis for the patient presented, therefore, is better than one usually anticipates.

DR. S. EPSTEIN, Marshfield, Wis.: This case certainly presents a difficult therapeutic problem. I agree with Dr. Montgomery that the histologic examination shows a squamous cell epithelioma of relatively low grade malignancy. One would not expect it to extend too much in depth. Application of an appropriate amount of roentgen or radium radiation to an epithelioma extending all around the anus may lead to a rather distressing and painful reaction around the rectum. I suggest removal of the tumor with the electric loop after a colostomy has been performed.

DR. L. A. BRUNSTING, Rochester: I believe that roentgen therapy is the method of choice in this case.

Livedo Reticularis. Presented by DR. S. E. SWEITZER, Minneapolis.

I. K., a white woman aged 21 years, was first seen at the dermatologic clinic of Minneapolis General Hospital on Sept. 28, 1943, when she complained of cold clammy feet and a purplish mottling of the skin of the legs. She stated that the eruption had been present all her life. She gave a history of a congenital patent interventricular septum and tricuspid insufficiency. At the age of 2 years she suffered from poliomyelitis.

Examination reveals a young woman of small stature and poorly nourished. Her feet are cold and clammy, and there is a livid reticulation of the knees and legs.

The Wassermann reaction was negative.

DISCUSSION

DR. L. A. BRUNSTING, Rochester: Livedo reticularis is a peculiar disease characterized by obliteration of the smaller arterioles of the extremities, particularly the legs, and there is a compensatory dilatation of the superficial vessels to give the mottled appearance of the skin. Sympathectomy increases the peripheral blood flow and brings about symptomatic benefit that is worthwhile. For patients with milder involvement the same benefit can be obtained by a change to a region of low atmospheric pressure, such as a high altitude affords. Recently I saw a young woman with this disease who had superficial ulcerations of the lower part of the legs. She moved from Omaha to Cheyenne, Wyo., and thereafter was free from symptoms for seven or eight years, but eventually there was a recurrence and sympathectomy was performed.

Lichen Planus Ocreaformis. Presented by DR. S. E. SWEITZER, Minneapolis.

S. K., a white man aged 87, was seen at Minneapolis General Hospital in 1941, when he complained of purplish spots on his legs. The spots had been present for eight years, and during that time they had slowly increased in number. There was pain in the knees, ankles and feet. He was seen again in September 1943, at which time he complained of itching of the forearms. Topical applications quickly cleared this dermatitis. The mottled appearance of the skin of the legs, which was noted in 1941, was still present though asymptomatic.

The Wassermann reaction was negative.

Examination reveals a well developed, somewhat overnourished old man. A purplish brown painless papular eruption covers the lower part of the legs from the ankles to a point a few centimeters below the knees. There is a scale adherent to the individual lesions, which are diffusely scattered. The lesions are sharply circumscribed and indurated and slightly elevated and dry.

DISCUSSION

DR. S. E. SWEITZER, Minneapolis: None of us could make a definite diagnosis when we first saw the patient. Clinically we thought the eruption looked like lichen planus. We never were able to confirm this diagnosis microscopically until we performed this most recent biopsy.

DR. H. E. MICHELSON, Minneapolis: There has been a great deal of discussion about the term "ocreaformis."

It is hard for me to decide just precisely what it means. For this particular patient I am unable to make a diagnosis.

DR. H. MONTGOMERY, Rochester: There is nothing in the histologic changes to suggest lichen planus. Deposition of hemosiderin and the mild degree of infiltrate suggested the possibility of purpuric pigmented lichenoid dermatitis of Gougerot and Blum.

Healed Scleroderma. Presented by DR. S. E. SWEITZER, Minneapolis.

S. B., a white woman, aged 54, in April 1937 first noted itching of the skin of the shoulders and thickening of the skin of the thighs and shoulders. This spread so that by February 1938 the entire back and abdomen and the lower part of the chest were involved. The cutaneous lesions were leathery, indurated tense plaques, ivory-colored and sharply demarcated from the surrounding normal tissue. Treatment consisted of six sessions of fever therapy of four hours each at 104 F. Since this treatment there has been a slow regression of the disease until the present time.

The patient is a well developed white woman with blotchy pigmented areas on the chest, abdomen, back and thighs. There is an indurated white area 2 by 4 cm. on each breast inferior to the nipple.

DISCUSSION

DR. S. E. SWEITZER, Minneapolis: This patient is shown today to demonstrate what can be done in treating this disease.

DR. L. A. BRUNSTING, Rochester: There seems to be no question in this case of the favorable influence of the fever treatment on the scleroderma, and such treatment is worth while. The disease has its spontaneous ups and downs, however, and I have seen relapses after fever treatment.

Circumscribed Scleroderma. Presented by DR. H. E. MICHELSON, Minneapolis.

D. S., a white girl aged 10 years, noted a red swelling on the lateral aspect of her left foot following a bruise a year ago. Without treatment the lesion subsequently decreased in size but did not disappear. Six months ago it became pale and thick and began to increase in size. Three months ago a similar, though smaller, lesion appeared just distal to the first, and it likewise increased slowly in size.

During the fall, winter and spring months for the past four years the patient has been given sulfanilamide for rheumatic heart disease.

Examination shows two irregular plaques on the lateral aspect of the left foot, one 2.5 by 3.5 cm. and the other about 1 cm. in diameter. They are firm, thickened and ivory colored with a dusky red border.

DISCUSSION

DR. C. W. LAYMON, Minneapolis: It is interesting to note that the child was first seen in the pediatric department and was referred to plastic surgeons for skin grafting. They were ready to perform the operation before a diagnosis was made, when, as a side thought, the child was sent to the dermatologic clinic.

Erythema Palmare. Presented by DR. S. E. SWEITZER, Minneapolis.

J. C., a 28 year old white woman, first noticed reddening of the palms in the third month of her first pregnancy. The erythema persisted through the subsequent two years. There was no evidence of pulmonary or cardiac disease.

Examination shows a passive hyperemia of both palms without tenderness or pain.

Erythema Palmare. Presented by DR. L. H. WINER, Minneapolis.

J. R., a white woman aged 32 years, complained of itching and redness of the palms which occurred during the seventh month of her first pregnancy, in 1941. The disease disappeared after delivery. In the seventh month of the subsequent pregnancy, in 1942, the erythema reappeared on the palms and persisted until the present time, two months after delivery. This erythema spread to involve the paronychial areas of the fingers.

The Wassermann reaction was negative.

Examination discloses a passive hyperemia and increased moisture of both palms. There is no tenderness or pain.

DISCUSSION OF CASES OF ERYTHEMA PALMARE

DR. JOHN MADDEN, St. Paul: During the past years I have seen several patients with a similar disease hospitalized for pulmonary tuberculosis. Shortly after they became ambulant the erythema disappeared. Biopsies showed no abnormality. The patients did not respond to any form of treatment while they were in the hospital.

DR. S. E. SWEITZER, Minneapolis: I think the disease is more often associated with pregnancy than with tuberculosis.

DR. L. A. BRUNSTING, Rochester: I have found this disease not uncommonly in males.

Subungual Exostosis. Presented by DR. S. E. SWEITZER, Minneapolis.

L. L., a 59 year old white woman, was seen in the outpatient department of Minneapolis General Hospital in the summer of 1943, when she complained of a painful warty growth under the nail of the left great toe. The growth, which had been present for three months, appeared to be a foreign body. It was removed, and the pathologist reported it to be a verruca vulgaris. The toe was treated with roentgen rays, but relief was not obtained. A roentgenogram of the toe was then made.

Examination shows an elderly woman with a warty growth under the nail of the left great toe. The lesion is painful on pressure.

DISCUSSION

DR. S. E. SWEITZER, Minneapolis: This woman complained of a great amount of pain. We confirmed our diagnosis with a roentgenogram of the toe. Surgical removal is indicated.

DR. S. EPSTEIN, Marshfield, Wis.: Such growths seem rare. I remember one identical to the one shown, also involving the great toe. The lesion appeared to be a wart. The first roentgenogram of the toe failed to show the exostosis because it was taken only in the anteroposterior plane.

Lichen Chronicus Simplex Associated with Hypoestrogenic Keratoderma. Presented by DR. S. E. SWEITZER, Minneapolis.

E. M., a 67 year old white woman, first came to the dermatologic clinic of Minneapolis General Hospital in July 1943. She stated that she had had a patch of thickened skin on the right elbow eight years previously.

Three months before her admission to the clinic she noted pruritic lesions on the knees and elbows. On examination there were sharply circumscribed lichenified brick red lesions. Hyperkeratosis and fissuring of the soles were also present. Treatment consisted of daily doses of 1 mg. of diethylstilbesterol for one month and applications of petrolatum topically. At weekly intervals for the past three weeks she has been given roentgen treatments of 150 r each.

The patient presents sharply circumscribed lichenified brick red plaques on the prepatellar surfaces and on the olecranons. The soles are now normal and painless.

The histologic sections showed hyperkeratosis and acanthosis with slight clubbing of the epidermal pegs. There is intense edema, with microscopic vesicle formation in several of the papillary bodies. The papillary layer is moderately edematous throughout, with a diffuse dense lymphocytic infiltrate. Except for moderate edema the reticular layer is uninvolved.

DISCUSSION

DR. L. A. BRUNSTING, Rochester: What has been your experience in regard to the return of menstrual bleeding as a result of estrogenic therapy?

DR. F. W. LYNCH, St. Paul: There has been raised in this case the question of differentiation from psoriasis. Certainly, similar scaling plaques on the elbows and knees are frequently regarded as psoriatic, though points of difference are present. In psoriasis one would expect the papules to be more sharply set off from the normal skin, the scale to be gray or white rather than yellow or brown, the underlying inflammation to be more red than brown, the lesions to be grouped over the elbows and knees rather than restricted to the proximal ulnar and tibial prominences and the itching to be less severe. The characteristic histologic changes of psoriasis are not present in the sections presented tonight.

I believe that if one is unwilling to regard the palmar and plantar eruption as a form of neurodermatitis there is still insufficient evidence to support a diagnosis of psoriasis.

When uterine bleeding develops in the course of estrogenic therapy there is a difference of opinion as to the proper course. Many gynecologists advocate temporarily stopping treatment, resuming it with small dosage after symptoms have left. Others believe that within the age group particularly susceptible to cancer it is best not only to stop the treatment but to curet the uterine mucosa in order to determine the presence or absence of coincidental cancer. Few, if any, believe that estrogenic therapy causes cancer in previously normal women.

Scleredema Adultorum. Presented by DR. C. W. Laymon, Minneapolis.

E. H., a white woman aged 42, noted painful swellings of the abdominal skin in July 1943. The skin on the nape of the neck, back, breasts, face, arms, buttocks and thighs was likewise affected. The dermatitis appeared after an infection of the respiratory tract and sinusitis. Within one month after the first attack almost complete involution of the induration occurred. Two weeks ago the patient had another infection of the respiratory tract and the same symptoms recurred.

The face, chest, nape of the neck, back, abdomen, buttocks, thighs and arms are involved. The hands, feet and mucosae are free. There are no surface or color changes. The affected parts are infiltrated as though with paraffin.

DISCUSSION

DR. L. A. BRUNSTING, Rochester: Recurrence of scleredema may happen. I remember 1 case of the disease in a woman in which there was a lapse of several years between the original episode and the recurrence. Fortunately, the disease is self limited, and there are no appreciable sequelae.

DR. C. W. LAYMON, Minneapolis: There are some unusual features about this case. The process started on the abdomen instead of on the nape of the neck. The nodules were painful, which is not the rule. Involution occurs within a few months in the majority of cases; in this patient complete involution occurred within four weeks, but lesions recurred shortly thereafter.

A Case for Diagnosis (Lupus Erythematosus? Dermatitis Factitia?). Presented by DR. H. E. MICHELSON, Minneapolis.

A. S., a white woman aged 61 years, noted a crusted lesion on the scalp three and a half years ago. A year later blisters appeared behind the left ear and on both cheeks. Shortly thereafter lesions developed in her mouth. Though the lesions would at times almost heal, the general trend, despite treatment, was of increasing severity and extension.

On examination sharply marginated lesions are noted on each cheek and behind the left ear. One small lesion is present behind the right ear and another on the forehead. All lesions are pink and atrophic and are covered with a thin gray easily removed scale. There are telangiectatic vessels and brown crusts on lesions of the cheeks. There is mild ectropion of the left lower lid adjacent to the upper border of the lesion of the left cheek. There is a large round bald area on the dome of the scalp with a central eroded portion and thin atrophic skin at the margins in which small islands of normal skin are seen. Eroded lesions, irregular in shape and covered with a thin gray film on a red base, are present on the roof of the mouth.

DISCUSSION

DR. H. E. MICHELSON, Minneapolis: In this case diagnoses of lupus erythematosus, pemphigus and a self-induced eruption were considered. The more I observe the patient the more I favor lupus erythematosus. There is little secretion for the size of the lesions. The woman herself is interesting. She has consulted many physicians and stays with each one only a short time. She never goes to the physician in her home town. It will be hard to keep her in the hospital, but I think she should be hospitalized for observation.

DR. L. A. BRUNSTING, Rochester: I favor the diagnosis of disseminated discoid lupus erythematosus, although the moist plaque involving so much of the scalp is decidedly unusual. The pattern of the eruption on the skin and the lesions on the mouth do not seem to fit in with dermatitis factitia.

DR. F. W. LYNCH, St. Paul: Many months ago, when I first saw this patient, I thought she had lupus erythematosus. I now feel less sure of the diagnosis, though some features still point in that direction. I think she should be placed in the hospital for observation in order to rule out the possibility of her eruption being self produced.

Dr. H. Montgomery, Rochester: The intactness of the epidermis, without parakeratosis and with preservation of the stratum granulosum, together with subepidermal bullae and liquefaction of the basal layer suggests the possibility of a bullous lupus erythematosus. Histologic changes did not fit in with factitial dermatitis or with a bullous form of lymphoblastoma.

LOS ANGELES DERMATOLOGICAL SOCIETY

William H. Goeckerman, M.D., *Chairman*

Clement E. Counter, M.D., *Secretary*

Oct. 12, 1943

Rosacea-Like Tuberculid of Lewandowsky. Presented by Dr. A. Fletcher Hall.

J. E. C., a man aged 31, an aircraft assemblyman, began to notice "pimples" on his upper lip in February 1943. During the following two months similar lesions appeared on the cheeks and forehead. All lesions have been symptomless, and none have suppurated. In August 1943 the lesions were noninflammatory. Some were red, and others were yellowish pink. Now no lesions are red; many are skin colored, and others are of a light tan color. All lesions are slightly elevated above the level. Diascopy reveals apple jelly pigmentation in the center of some of the lesions. All lesions had this appearance on diascopic examination before treatment. The result of a tuberculin patch test in April 1943 was positive. A roentgenogram of the chest showed evidence of glandular involvement. Examination of the sputum did reveal tubercle bacilli. A specimen for biopsy, taken by a Los Angeles dermatologist, is reported to have shown chronic inflammation. Intramuscular injections of a bismuth preparation by another dermatologist are reported to have provoked reactions of fever, with no apparent improvement. Ten weekly intravenous injections of gold sodium thiosulfate (progressing by 5 mg. per week from 5 to 50 mg.) were given. Intramuscular injections of crude liver extract in doses of 2 cc. (2 units per cubic centimeter) were given twice weekly contemporaneously. Steady improvement has occurred.

DISCUSSION

Dr. Chris Halloran: I agree with the diagnosis and also approve of Dr. Hall's method of treatment. This disease responds well to gold sodium thiosulfate.

Dr. L. F. X. Wilhelm: I also am using crude liver extract along with intravenous gold therapy in cases in which gold therapy is indicated.

Dr. H. C. L. Lindsay: I think an eruption like this does well when other treatment is supplemented by ultraviolet irradiation from a water-cooled quartz mercury vapor arc lamp with pressure (contact treatment).

Dr. Samuel Ayres Jr.: I was not altogether satisfied with the diagnosis. From all I could see, the eruption corresponds to rosacea. I should not make a diagnosis of rosaceaform tuberculid without a biopsy.

Dr. Saul Robinson: It seemed to me that the lesions looked like tuberculosis miliaris disseminata faciei rather than rosaceaform tuberculid. These were papular lesions, and on pressure they presented a brownish center, as is seen in tuberculosis miliaris faciei.

Dr. Nelson P. Anderson: I agree with Dr. Robinson. I think that this eruption probably is tuberculosis miliaris disseminata faciei.

Dr. Kenneth Stout: I thought the typical lesion was a smooth waxy nodule. I believe the diagnosis is epithelioma adenoides cysticum.

Dr. A. Fletcher Hall: When I selected a title for this case I hesitated between the title under which it was presented and tuberculosis miliaris faciei. I chose the title I did because in looking through recent copies of the "Year Book of Dermatology and Syphilology" it seemed that most of the cases were described as rosacea-like tuberculids rather than tuberculosis miliaris faciei. Becker and Obermayer's text considers the two terms synonymous. I have been at a loss to know where to draw the line between them. I think it is not rosacea because of the distribution and because of the lack of erythematous reaction around the lesions and because the apple jelly appearance can be easily demonstrated, especially in lesions around the glabella. I think it is not an epithelioma adenoides cysticum because of its response to gold and liver therapy.

Dermatitis Herpetiformis Limited to the Scalp. Presented by Dr. Samuel Ayres Jr.

A. E., a man aged 54, has been suffering for seven months with an intensely itchy scalp. Lesions occurring on the scalp were described as blebs which were easily broken. A proprietary hair tonic had been used weekly for several years, but its elimination has made no change in lesions. In July 1943 the eruption was limited to the top part of the scalp and consisted of numerous superficial pinhead-sized to dime-sized superficial excoriations and crusts. Also present in the same area were smooth white atrophic nickel-sized patches resembling pseudopelade except that they were surrounded by excoriations. In September 1943 he presented two split pea–sized superficial blebs located just within the hair line toward the front of the scalp. Both contained straw-colored fluid. A smear from a fresh bleb revealed no bacteria. A culture was sterile. Wright's stain of fluid from a bleb revealed many polymorphonuclear leukocytes and eosinophils. Treatment has consisted of seven doses of roentgen rays varying from 1/6 to 1/8 skin unit. Ten per cent boric acid ointment has been applied locally. In September 1943 the patient was given nicotinamide (300 mg. daily). The eruption has improved somewhat, but new blebs still form from time to time.

DISCUSSION

Dr. Saul Robinson: I believe that this is lupus erythematosus of the scalp, in spite of the crusting.

Dr. Chris Halloran: I observed atrophy in the center of the lesions and an active suppurative process in the hair follicles of the borders. I favor a diagnosis of folliculitis decalvans.

Dr. A. Fletcher Hall: I agree with Dr. Robinson. The lesion on the top of the head particularly, which showed alopecia, was suggestive of lupus erythematosus, showing follicular stippling around the edges and what appeared to me to be atrophic scarring in the center with loss of hair. I think lupus erythematosus can present bullous elements, and the fact that there are bullae does not rule out that diagnosis.

Dr. Kendal Frost: This is a puzzling case. There have been presented before this society on previous occasions 1 or 2 patients with pustular eruptions of the scalp which have been peculiarly resistant to any sort

of treatment. This patient demonstrated one lesion on his scalp that had ruptured shortly before he came to the meeting. This lesion was either a vesicle or a pustule, the presence of which rules out lupus erythematosus according to my conception of that disease. There are types of folliculitis which could have produced the amount of alopecia present.

DR. SAMUEL AYRES JR.: I do not think this is a case of lupus erythematosus. I was unable to convince myself that there is any stippling present such as is found in this disease. The lesions in this case are intensely pruritic. The only primary lesions I have seen have been small (pea-sized) clear blebs, almost pemphigoid but smaller, which break rather easily. They are not a follicular type of lesion. Wright's stain of fluid from a bleb showed leukocytes and large numbers of eosinophils. I have not the remotest idea what this disease is, but in presenting the case I thought that dermatitis herpetiformis might be the most likely possibility.

Subacute Disseminate Lupus Erythematosus. Presented by DR. JOHN D. ROGERS.

A. V., a woman aged 64, began to have a dry red scaly eruption on the face, on the forearms and on the upper part of the chest six months ago. The first lesions appeared on the left cheek. Subjective symptoms have been lacking from the onset. The general health has been good. An irregular erythematous eruption is present on the cheeks, nose and lower eyelids. There is considerable edema of the eyelids. There are no atrophic lesions on the face. The forearms present irregular scaly papular lesions which have prominent margins and centers showing some atrophy. Similar lesions are scattered over the backs of the hands and on the upper part of the chest. A general physical examination revealed chronic infected tonsils and much pyorrhea, with several abscessed teeth. The sedimentation rate is 50. Slight improvement followed the giving of eight intramuscular injections of bismuth subsalicylate in oil. These injections were discontinued because of dark discoloration in the vicinity of the bad teeth. Afterward four weekly intravenous injections of gold sodium thiosulfate were given, and this treatment seemed to aggravate the eruption. The teeth are being extracted.

DISCUSSION

(All agreed with the diagnosis as presented.)

Contact Dermatitis Due to Hair Lacquer. Presented by DR. IRVING BANCROFT.

E. F., a woman aged 42, applied a cosmetic preparation (hair lacquer) to the hair, and three days later an erythematous itching eruption developed on the face and arms. It elicited a positive reaction when applied as a patch test. At present the eruption is disappearing. An ointment of solution of aluminum acetate in an oxycholesterol-petrolatum base (aquaphor) has been the only medicament used.

DISCUSSION

DR. ANKER JENSEN: I have a patient with a similar contact dermatitis caused by the same hair lacquer.

DR. SAMUEL AYRES JR.: I had a patient today who had used lacquer pads on her hair. She is just recovering from the same type of eruption.

DR. IRVING BANCROFT: This hair lacquer is being investigated. I do not yet know the chemical which causes the eruption.

Colloid Degeneration of the Skin (Colloid Milium). Presented by DR. SAUL S. ROBINSON.

P. S., a man aged 45, who operates a parking lot, has had a papular eruption on the backs of his hands for over ten years. The lesions on the cheeks have come more recently. Individual lesions are firm nodules which appear almost translucent and measure about 1 mm. in diameter. They tend to be in groups of eight to ten. Biopsy plainly shows areas of colloid degeneration in the upper part of the cutis. The Wassermann reaction was negative. Treatment has consisted of oral and intramuscular administration of ascorbic acid (vitamin C).

DISCUSSION

DR. CHRIS HALLORAN: I agree with the diagnosis of colloid pseudomilium. In this connection, attention is called to the report of a case by Arnold, with a review of the nomenclature. (ARCH. DERMAT. & SYPH. **48**:262-267 [Sept.] 1943.)

DR. SAMUEL AYRES JR.: Has the patient made any response to vitamin C therapy? I think it would be well to present this patient again in two or three months.

DR. SAUL ROBINSON: These cases of colloid degeneration of the skin are of interest because in nearly all of them the patients are persons who are exposed to the weather, including excess sunlight. The actinic rays of the sun must play a part in the degeneration. Since Dr. Nelson P. Anderson introduced vitamin C therapy in treatment of this type of disease, Dr. Way, of San Francisco, has presented the case of a patient whom he treated for one year with ascorbic acid and whose lesions disappeared.

Sporotrichosis. Presented by DR. SAMUEL AYRES JR.

J. A., a man aged 60, has had the present lesion for the past five months. It began with a single lesion on the dorsum of the left fourth finger immediately after a traumatic abrasion on that finger. The patient was working with moldy redwood at the time of the onset. It had been several years since he had been out of California.

The eruption appears entirely on the extensor surface of left fourth finger, the left hand and the left forearm to the elbow. Individual lesions are in groups and are nickel-sized dull red inflammatory nodules with a tendency to a linear or a circinate form. Most of the lesions show ulceration. There is a partly healed arciform group of nodules on the affected forearm just below the elbow. Kahn and Kline tests gave negative reactions. A culture on Sabouraud's medium showed a smooth gray growth with a central piling-up as well as a downward extension into the medium. Microscopic appearance of a mount of the culture was characteristic of Sporotrichum schenki. Biopsy revealed a granulomatous process consistent with sporotrichosis. Involving the cutis to a deep level were circumscribed areas composed of three zones. The central zone was of necrosis with polymorphonuclear leukocytes. The second zone included epithelioid cells and Langhans giant cells. The outer zone was made up of dense lymphatic infiltration. Treatment has included administration of potassium iodide by mouth. Ingestion of 30 drops a day was begun on Sept. 17, 1943. This was increased by 5 drops each day until the patient was taking 180 drops a day. He is now maintaining that large dosage and his condition is much improved.

DR. H. P. JACOBSON: Clinically this is a tuberculoid form of sporotrichosis. As far as I can recall, this is the first authentic case of sporotrichosis in which the

infection was contracted in California. There have been about 60 cases of the disease reported in the American literature. I have seen 3 other patients with sporotrichosis, and the infection in none of them had originated in this state. The culture demonstrated by Dr. Ayres tonight is characteristic of the growth of the causative organism of sporotrichosis.

DR. SAMUEL AYRES JR.: This is the first case of sporotrichosis I have encountered in private practice, and it originated in California.

Lupus Vulgaris. Presented by DR. ANKER JENSEN.

T. W., a Chinese woman aged 44, had a small pimple on the left cheek about three years ago. It has gradually enlarged to the present size and shape of a half-dollar. Its margin is a red papular border. The central part of the lesion is a smooth atrophic scar. Diascopic pressure apparently produces apple jelly nodules at intervals in the papular margin.

DISCUSSION

DR. A. FLETCHER HALL: I favor the diagnosis of granuloma annulare.

DR. NELSON P. ANDERSON: I should like to see if the diagnosis of lupus vulgaris can be substantiated by a biopsy. I doubt it. In the first place, if Dr. Jensen's diagnosis is correct, it is indeed a rather large patch to develop in three years. Secondly, the lesion has a clearing center with a raised peripheral nodular border and an entire absence of tubercles in the smooth central part. Thirdly, the patient is Chinese. My clinical diagnosis is leprosy. It does not exactly fit the picture presented by the history, but these Chinese are clever people and they are prone to like every diagnosis except leprosy. I shall not be a bit surprised to find that on further study this disease will prove to be a sarcoid type of leprosy.

DR. SAMUEL AYRES JR.: When I saw the patient, my only thought was lupus vulgaris. I thought the lesion looked rather characteristic. I agree with all that Dr. Anderson has said, however, and I think a biopsy would solve the problem.

DR. SAUL S. ROBINSON: If biopsy proves the lesion to be lupus vulgaris, plastic surgical repair should be considered. A plastic surgical operation was performed on a patient of mine who had a small lupus vulgaris lesion two years ago, and the result has been cosmetically successful.

DR. ANKER JENSEN: I had just seen this patient for the first time a few minutes before the meeting, and I thought it would be interesting to show her, inasmuch as one encounters so few cases of lupus vulgaris. A biopsy will be made and laboratory tests performed.

Localized Scleroderma. Presented by DR. SAUL S. ROBINSON.

F. C., a man aged 53, has had the present lesions for the past six years. He states that the patches begin as hard firm red areas which later become pigmented and whitened. At present there are about six hand-sized light brown pigmented and erythematous plaques on the trunk and extremities. All lesions are oval. None are firm now. The basal metabolic rate is normal; the Wassermann test elicited a negative reaction, and the blood count is normal.

DISCUSSION

DR. SAMUEL AYRES JR.: I do not see why one should make a diagnosis of scleroderma when there is no evidence of hardening of the skin. I favor a diagnosis of macular atrophy. It is true that many of the lesions have a violaceous tint.

DR. CHRIS HALLORAN: I agree with the diagnosis of morphea, but I did not feel any induration in any of the lesions. The patient stated to me that none of the lesions were firm at any time.

DR. SAUL S. ROBINSON: A biopsy performed by Dr. Franklin Ball some time ago was reported to have shown scleroderma. As time goes on, the lesions become softer and slightly pigmented.

Pemphigus Erythematosus (Senear-Usher). Presented (for the second time) by DR. CLEMENT E. COUNTER.

V. S., a man aged 53, has suffered from a localized eruption on the left cheek for the last eighteen months. The first lesion noted was a small blister on a slightly red base. It was about 2 cm. anterior to the left ear. The blister soon ruptured. The first physician consulted lightly electrodesiccated the lesion. When it did not heal, the patient was referred to a surgeon who makes a specialty of treating malignant tumors. He gave 6,000 r of roentgen therapy localized to a dime-sized area. When the reaction to this treatment had faded, a rim of smooth red papular eruption began to extend beyond the area so treated. Blisters continued to form on this ever enlarging lesion until April 1943, when the patient was presented before this society. The diagnosis under which he is presented tonight was suggested. Gold sodium thiosulfate therapy was begun, and the lesion has become smaller. Vesicles have been much fewer.

A fading red oval patch about 5 by 8 cm. just anterior to the left ear is the only lesion present. Near its middle is a circular patch of atrophy having no hair. General examination has failed to reveal any hidden foci of infection. The Wassermann test elicited a negative reaction in December 1942. At the same time further hematologic studies revealed a hemoglobin content of 15.8 Gm., 5,040,000 erythrocytes and 7,700 leukocytes, with neutrophils 63 per cent, lymphocytes 31 per cent, monocytes 3 per cent and eosinophils 3 per cent. A biopsy in July 1943 revealed cellular infiltration in the cutis which was consistent with the Senear-Usher syndrome.

In the last seven months fifteen intravenous injections of gold sodium thiosulfate have been given at weekly intervals. The first dose was 10 mg., and subsequent doses were increased by 5 mg. until a maximum of 50 mg. was reached.

DISCUSSION

DR. A. FLETCHER HALL: I hazarded the diagnosis of pemphigus erythematosus when this patient was presented before, and I am still of the same opinion.

DR. CLEMENT E. COUNTER: This man was presented tonight to show the results of gold sodium thiosulfate therapy. He is much improved. I believe the good results of this therapy emphasize the diagnosis suggested by Dr. Hall and agreed to by many of the members at the meeting in April 1943.

Multiple Ulcerations of the Legs. Presented by DR. SAMUEL AYRES JR.

E. G., a man aged 56, who was presented for the second time tonight, was presented at the February 1943 meeting of this society (ARCH. DERMAT. & SYPH. **48**:327 [Sept.] 1943). The present eruption has existed since May 1942, when what was believed to be a vari-

cose ulcer on the left leg developed. Injections were made into some varicose veins, and more lesions appeared at the sites of injection. The patient was then thought to have blastomycosis and was given potassium iodide by mouth. Sulfathiazole was used locally. He was also given bromides while he was in the hospital, but this was before October 1942, when he came under my care. His treatment then included wet dressings of water containing zinc and copper sulfate (Alibour water), 2 per cent aqueous solution of gentian violet (methylrosaniline) and chlorohydroxyquinoline ointment. After about six weeks of this treatment he was discharged from the hospital about 99 per cent well. Since then the patient has acquired intolerance to chlorohydroxyquinoline and the lesions have recurred, but they have never been as severe as when first examined. In spite of negative Kahn and Kline reactions, his treatment has included mapharsen and a bismuth preparation on the basis of his having had syphilis years ago. Other medicaments which were applied locally were 5 per cent sulfathiazole ointment, 5 per cent sulfadiazine ointment and tyrothricin (20 to 40 mg. per hundred cubic centimeters) in an ointment and in a solution applied as a wet dressing. These treatments were paralleled by a long series of intravenous injections of autogenous vaccine containing Staphylococcus albus. Repeated cultures, on aerobic and on anaerobic mediums, on Sabouraud's medium and on blood agar have failed to show any other organisms than Staph. albus. A biopsy in March 1943 showed a chronic inflammatory process suggestive of an iodine eruption. This was at a time when the patient had not had iodide therapy for six months.

DISCUSSION

DR. H. P. JACOBSON: That this man has tertiary syphilis is still my opinion, as it was when he was first presented. The ulcerations, while multiple and involving both lower extremities, nevertheless show suggestive outlines and are interspersed with fairly suggestive syphilitic scars. The patient's treatment under Dr. Ayres has included an arsenical and a bismuth preparation for a portion of the time since he was last presented here. It is my opinion that only after these antisyphilitic treatments were stopped did the ulcerations become worse.

DR. SAUL S. ROBINSON: I agree with Dr. Jacobson that this is a case of tertiary syphilis although the serologic tests are negative. I think that if fever therapy were used along with injections of arsenicals and bismuth preparations and high vitamin preparations were given improvement would be achieved.

DR. H. C. L. LINDSAY: The disease appears to be tertiary syphilis. I suggest that a study of the spinal fluid be made.

DR. CHRIS HALLORAN: How much specific therapy for syphilis has this man had?

DR. SAMUEL AYRES JR.: He has had about fifteen injections of mapharsen and about the same number of injections of bismuth.

DR. CHRIS HALLORAN: I think that it is not syphilis but that it is an ioderma or a bromoderma.

DR. L. F. X. WILHELM: The fact that the lesions are bilateral should exclude tertiary syphilis.

DR. IRVING BANCROFT: I do not think that it is syphilis.

DR. ROGER WAKEFIELD: When I talked with the man alone he wanted to know what I thought about the taking of bromides for headaches, as if he were now using bromides occasionally for the relief of headaches.

DR. SAMUEL AYRES JR.: I must be a pretty poor detective, for my first diagnosis was an eruption due to a bromide. To the best of my knowledge he has received neither iodides nor bromides for nearly a year unless, as Dr. Wakefield suggests, he has been taking them on the side. The remarks of Dr. Jacobson that there was apparent clearing while antisyphilitic treatment was being used is not exactly correct. Even though he was much improved on leaving the hospital his antisyphilitic therapy was continued, but at about the same time an idiosyncrasy to chlorohydroxyquinoline developed which necessitated its discontinuance and then it was that the eruption relapsed.

Lupus Erythematosus Complicated by Dermatitis Factitia. Presented by DR. SAMUEL AYRES JR.

P. P., a woman aged 21, has had an eruption on her face for the last four years. The lesions on the arms have appeared more recently. She says that she has not excoriated the lesions, but she admits that she is nervous. She has occasional attacks of arthritis in her hands. Her tonsils were removed at the age of 10 years.

The patient appears somewhat nervous. There are numerous lesions on the face, with others scattered over the upper part of the back and the upper outer aspect of the arms. Individual lesions are typical of the discoid type of lupus erythematosus, with areas of redness showing a slight amount of grayish roughness, scaling and pronounced follicular stippling. Lesions are confluent across the nose. There is a large area of depressed scar on the left side of the forehead including a portion of the left eyebrow. There are other lesions with depressed central scarring on the cheeks and the arms. Various lesions on the cheeks and arms exhibit, in addition, deeply ulcerated and crusted areas. The eyes show increased vascularization of the cornea in the palpebral opening.

Conditions revealed on roentgen study of the chest about six months ago are said to have been normal. Kahn and Kline tests elicited negative reactions in July 1943. A faint trace of albumin was discovered in the urine along with occasional erythrocytes and a few pus cells. A blood count showed 77 per cent hemoglobin, 4,240,000 erythrocytes (color index 0.9) and 12,300 leukocytes, of which 46 per cent were neutrophils, 54 per cent lymphocytes and 5 per cent monocytes.

Treatment has included a general vitamin tonic by mouth, and instructions about the necessity of not tampering with the lesions. She has received nine intramuscular injections of bismuth subsalicylate in oil (2 grains [0.13 Gm.] of the salt in each dose) and four intravenous injections of gold sodium thiosulfate (increased from 5 mg. at first to 20 mg. per dose). A 10 per cent boric acid ointment was prescribed for local use. The lesions have tended to become worse rather than to improve.

DISCUSSION

DR. A. FLETCHER HALL: I should like to ask if there has been any successful attempt to find out what the patient is using to produce the factitial part of the eruption. Has an attempt been made to get her to consult a psychiatrist? Such a person should be under the care of a competent psychiatrist. I think her main problem is not lupus erythematosus but a major psychosis.

DR. SAUL S. ROBINSON: I have never seen any benefit result from psychiatric consultations for such patients. I wonder if any one has had any success with them.

DR. ANKER JENSEN: Three years ago I sent a patient to a psychiatrist. Her husband and then her son had died in quick succession, and she was left alone in the world. Three months later, when I first saw her, she had neurotic excoriations with serious ulcerations on the side of her neck. She was introduced to a psychiatrist, and he got her straightened out promptly; she still comes in at intervals to thank me for referring her to the psychiatrist.

DR. SAMUEL AYRES JR.: This patient lives at a distance, and, although I have had psychiatric consultation in mind, I have not arranged for it yet because of her infrequent visits. She stoutly maintains that she does not manipulate the lesions and insists that they develop spontaneously, which is, of course, an old story. She always appears nervous and ill tempered. Probably there is a strong psychic factor. This is perhaps easier for me to understand after seeing the husband tonight for the first time. Obviously he has an inadequate personality.

Hodgkin's Disease with Cutaneous Manifestations. Presented by DR. SAMUEL AYRES JR.

H. H., a woman aged 68, has had the present eruption for the last ten years. There is no family history of tuberculosis. The patient had malaria and typhoid many years ago. The itching eruption started on the arms and soon became generalized. There has been swelling of the left forearm for the past two years. Roentgen therapy was given to the enlarged cervical glands when the patient was in the Los Angeles County Hospital two years ago. There was improvement of the glands, although there was no improvement of the eruption. Itching is severe. The patient is somewhat thin and pale. The skin reveals generalized dryness with slight thickening and roughening. There is freckle-like pigmentation. A pebbly appearance is especially well marked on the forearms and the legs. The left arm and hand are somewhat swollen. There are numerous scattered excoriations. Masses of enlarged glands are present in the left axilla and on the sides of the neck. No masses are felt on the breasts. A blood count on Oct. 1, 1943 showed: erythrocytes, 4,210,000; leukocytes, 6,800; hemoglobin content, 58 per cent; color index, 0.7; neutrophils, 27 per cent; monocytes, 6 per cent, and lymphocytes, 17 per cent. A diagnosis of Hodgkin's disease was made from a biopsy of cervical lymph nodes in 1941.

DISCUSSION

DR. SAUL ROBINSON: I should like to request Dr. Ayres to make a histologic examination of the cutaneous lesions if possible.

DR. SAMUEL AYRES JR.: I have not had time for a biopsy. This woman has Hodgkin's disease with probable toxic manifestations in the skin. The lymph nodes were palpable under her arms. There were also masses in her chest and some in her neck. She has a pronounced anemia, with 58 per cent hemoglobin. I thought it would be interesting to show a patient with this combination of symptoms.

Melanoma. Presented by DR. JOHN D. ROGERS.

E. N., a woman aged 39, has had a small dark brown lesion on the left side of her neck since birth. It is described by the patient as having been the size of a green pea and having had no appreciable change in size until about eighteen months ago. Since then it has increased to 1.6 cm. The margins of the lesion are not elevated, and they are brown rather than blue-black like the central part. There are numerous other brown nevi over other parts of the body.

DISCUSSION

DR. NELSON PAUL ANDERSON: I think the entire lesion should be removed widely and deeply, with resection of the regional lymph nodes, but I suspect it is even now too late to remove the process completely.

DR. CHRIS HALLORAN: I believe that with excision and cauterization of the base there is hope that there will be no metastasis.

DR. H. P. JACOBSON: The lesion is probably a melanomatous carcinoma. According to the patient this lesion has been showing evidence of growth recently; yet there is no definite evidence at this time that metastatic extension has taken place. Certainly there is no evidence of regional adenopathy, and the patient's general condition is good. In view of these facts, I cannot subscribe to the pessimistic conclusions regarding the prognosis expressed by other members. Surgical excision with the cautery, radically and yet judiciously performed with the patient under general anesthesia, offers a good prognosis in this case.

A Case for Diagnosis (Lymphoblastoma?). Presented by DR. SAMUEL AYRES JR. and DR. ANKER JENSEN.

A. R., a white man aged 66, was born in the United States and has worked as an electric power lineman. About fifteen years ago he began losing hair. In two years he had universal alopecia. Generalized pruritus began three years ago. Associated with it was a gradual enlargement of the ears, nose and chin. Small "blisters" are also reported by the patient to develop on the extremities; they soon become small abscesses and later crust over. The pruritus and the enlargement of parts of the face have been progressive. There has been a gradual loss of about 14 pounds (6.4 Kg.) in the last two years.

There is no family history of carcinoma, hypothyroidism or epilepsy. No other member of the family has had any eruption. There has been no asthma or hay fever.

There is a loss of all hair and an enlargement of the ears, the nose and the chin. The eyelids are edematous, and the face is erythematous, with areas of excoriation and weeping. The central portion of the face shows wrinkling. Crusts and excoriations are scattered over the arms, the chest and the legs. Primary lesions are not present now. The finger nails are pitted and discolored.

The pupils are equal in size and react equally to light and in accommodation. The ears are enlarged to about twice normal size and are tender. The nose has a chronic watery discharge. The lips are edematous. The lungs are clear. The heart is normal. The abdomen is not distended or otherwise abnormal. There are no deformities of the extremities.

Examinations of the blood reveal: erythrocytes, 3,970,000; leukocytes, 15,700; neutrophils, 42 per cent; lymphocytes, 42 per cent; eosinophils, 3 per cent; monocytes, 6 per cent; monoblasts, 7 per cent; hemoglobin, 12.8 Gm.; blood sugar, 80 mg. per hundred cubic centimeters; nonprotein nitrogen, 34 mg.; uric

acid, 3.6 mg. and calcium 10 mg. Two biopsies were performed, and the report read: "Both sections show essentially the same picture, namely that of deep epithelial cysts."

DISCUSSION

DR. KENDAL FROST: I believe this whole picture can be explained on the basis of a chronic inflammatory process with resultant fibrous proliferation, something of the sort which produces rhinophyma. However, I really believe it will turn out to be one of the lymphoblastomas.

DR. CHRIS HALLORAN: I do not know what to call it, but it impresses me as some form of lymph stasis. The ears have the appearance of elephantiasis. The whole picture must be due to extensive lymph stasis with subcutaneous fibrous growth.

DR. ROGER WAKEFIELD: This man is the most grotesque-looking person I have ever seen. I am wondering whether the dystrophy of the nails is part of the picture or whether these changes are due to a chronic infection around the nails.

DR. IRVING BANCROFT: It seems to me that leprosy should be considered.

DR. NELSON PAUL ANDERSON: I do not know what this disease is, but I feel it is a definite syndrome. First, complete alopecia developed, and later the enlargements of the eyebrows, nose, ears and chin occurred. It is hard for me to believe that a low grade infectious process would attack both ears and produce a symmetric result like this. Then, too, there are the changes of the nails, mentioned by Dr. Wakefield. I believe it belongs to a definite group, even though I cannot name it and I do not recall any description in the literature of a similar syndrome.

DR. A. FLETCHER HALL: Realizing that acromegaly is concerned with overgrowth of bone and not of soft tissue but seeing the enlargement of soft tissue in the same areas as those affected by acromegaly and noting the presence of total alopecia, I am forced to think that there is some pituitary basis for the whole condition. I believe there must be some endocrine imbalance, and my first suspicion would be aimed at the pituitary. I think roentgen studies of the skull may give a clue.

DR. SAMUEL AYRES JR.: When I first saw this man at the hospital, I thought only of the term "gargoylism." That term has already been used to designate another entity. Dr. Hall's suggestion of pituitary disease is well taken. Last week I ordered roentgenograms of the patient's skull. Then, too, the whole process may be a manifestation of a lymphoblastoma. Perhaps the hypertrophy results altogether from the long-continued scratching. The changes of the nails are possibly secondary to a chronic paronychia. The patient said that the fingers with thickened nails have been inflamed around the bases of the nails.

Linear Lymphatic Nevus. Presented by DR. H. C. L. LINDSAY.

P. B., a girl aged 18 months, exhibits a slightly raised lesion on the left side of the chest. It extends downward and forward from the left axilla. It is ribbon-like, being about 6 mm. wide by about 8 cm. long. A similar linear lesion is present on the lower part of the chest. No inflammation or induration is present. There has been no change in length or in contour during the twenty-six days it has been under observation.

DISCUSSION

DR. A. FLETCHER HALL: The lesions suggested a dermographic reaction to scratching. The parents said that scratching other parts of the body does not produce a similar reaction. I thought of the possibility of a cervical rib or of a scalenus anticus syndrome modifying the sympathetic supply to the area and thereby bringing forth a localized reaction.

DR. NELSON PAUL ANDERSON: I think Dr. Lindsay is on the right track. The disease probably belongs to the same group as the cases described by Senear and Caro (Senear, F. E., and Caro, M. R.: Lichen Striatus, ARCH DERMAT. & SYPH. **43**:116 [Jan.] 1941). This is a peculiar type of linear eruption which occurs in childhood and disappears spontaneously, leaving little or no trace. An attempt has been made to place the disease in the nevus group.

A Case for Diagnosis (Chronic Benign Pemphigus?). Presented by DR. SAMUEL AYRES JR.

M. C., a girl aged 6 years, was admitted to the dermatologic department of the White Memorial Hospital with a generalized bullous eruption of about six months' duration. She was referred from the departments of dermatology and pediatrics of Temple University School of Medicine, Philadelphia. While a patient there she had been treated for a disease diagnosed as erythema multiforme with a question of an extensive contact dermatitis. Secondary infection was controlled with sulfathiazole orally and topically. Lesions began to appear on the palm and soles. The first lesions were small clear vesicles.

At present there are many pinhead-sized to pea-sized clear vesicles, which are tense and not umbilicated. New lesions show no erythematous base. Older lesions show a pink halo surrounding the vesicle, and the contents become cloudy. Other, still older, lesions have ruptured, and their sites are covered with yellow crusts. Many lesions are arranged in annular or semiannular shapes. The distribution is generalized, but lesions are most numerous on the dorsa of the toes, on the fingers, shoulders, flexor surfaces of the arms, inner aspects of the thighs and around the mouth. There are no mucosal lesions. Areas where the lesions have disappeared showed mild scaling and some hyperpigmentation.

Wassermann and Kahn tests elicited negative reactions. A blood count revealed: erythrocytes 4,200,000, leukocytes 7,200 (neutrophils, 54 per cent; lymphocytes, 35 per cent; eosinophils, 11 per cent) and hemoglobin content 13.3 Gm. The urine was normal.

DISCUSSION

DR. CHRIS HALLORAN: I have never seen a patient so young with true pemphigus, but clinically the lesions look like those of the disease described by Ayres and Anderson and by the Hailey brothers, familial benign pemphigus. Usually those lesions are on the flexor surfaces of the neck and in the axillas, but in 1 patient I followed they were later scattered about as they are in this patient.

DR. KENNETH STOUT: May I add the diagnosis of bromoderma to the differential list of diagnoses?

DR. H. C. L. LINDSAY: This is a case of dermatitis herpetiformis.

DR. NELSON PAUL ANDERSON: This case belongs in the group of cases of a disease first described by John Bowen, of Boston. It can be called a benign pemphigus

of childhood. Somewhat later the disease was encountered in St. Louis, where in several cases it followed vaccination. I wonder if the eruption in this case followed vaccination. The most recent paper on this disease was by Tomlinson and Cameron (ARCH. DERMAT. & SYPH. **38**:555, 1938). I do not believe this case is an instance of the disease that Dr. Ayres and I described, and I do not believe it is a case of dermatitis herpetiformis. I do believe it belongs among the cases of a group of benign pemphigoid eruptions in childhood which have a fairly good prognosis.

Leukoderma Acquisitum Centrifugum. Presented by DR. H. C. L. LINDSAY.

C. S., a girl aged 9 years, since infancy has had an area on the right frontal region which would become red. Vitiligo later developed on this area and has been present nine months. Other patches of vitiligo have been present in the left axillary region and around a mole on the back for about the last three months. These lesions of acquired vitiligo have developed like an areola of milk-white skin beyond a centrally situated mole. Vitiligo also exists independent of moles on the forehead and under the left arm.

Treatment has consisted only of the administration of gold tribromide (Noxon Toomey formula) in 8 minim (0.5 cc.) doses diluted, by mouth three times a day.

DISCUSSION

DR. A. FLETCHER HALL: This case is a good example of leukoderma acquisitum centrifugum, and the response to gold therapy seems gratifying.

DR. SAMUEL AYRES JR.: I wonder why this could not be a case of ordinary vitiligo. There are a number of pigmented lesions on the forehead. One appeared to be in a spot of vitiligo, and the other vitiliginous areas did not include pigmented lesions. I did not see the ones on the back.

DR. SAUL ROBINSON: Such a condition occurs not only with nevi but with lichen planus and psoriasis. It should really not be classified as vitiligo. There is definite loss of pigment surrounding the lesions, but vitiligo is a loss of pigmentation not associated with nevi.

DR. H. C. L. LINDSAY: I have never given a gold preparation to a patient with leukoderma centrifugum before, and I was surprised at the rapid improvement from the oral administration of gold tribromide solution.

Book Reviews

Elements of Medical Mycology. By Jacob Hyams Swartz, M.D. Price $4.50. Pp. 179, with 78 illustrations. New York: Grune & Stratton, Publishers, 1943.

The purpose of this book is defined by its author as that of "familiarizing students and physicians with the terminology of mycology, with the more common diseases produced by fungi, and with the laboratory procedures employed in diagnosis of these."

The book (exclusive of pictures) contains about one hundred pages of text and is divided into nine chapters. The first two are devoted to the discussion of the origin and general morphology of fungi, their physiology, classification and relation to disease and the methods of diagnosis of fungous infections. The following five chapters contain the description of important pathogenic fungi causing superficial and deep mycoses and also of some of the usual nonpathogenic contaminants. The trichophytin test is discussed in the eighth chapter and the action of sulfanilamide and its derivatives on fungi in the last, ninth, chapter. The book ends with a glossary of mycologic terms and a table with a summary of the clinical and mycologic findings as related to pathogenic fungi.

The book contains valuable material in a concise and easily readable form. Instructions concerning treatment and prophylactic measures against ringworm are given in a practical manner, with many useful details. There are 78 illustrations, clinical photographs, photomicrographs and drawings. With a few exceptions they are original. Those illustrating the laboratory specimens of fungi in tissues and in culture mounts are clear and instructive. However, figure 20, a hair containing Microsporon, an important specimen from the practical standpoint, is not demonstrative enough, since it does not show distinctly the characteristic spore case of mosaic-like spores surrounding the hair. For the sake of comparison and differential diagnosis it would be desirable to add pictures of trichophyton fungi in their "endo" and "ecto" appearance. The clinical photographs, especially of the scalp and nails, are not as good as those of laboratory specimens.

Much space and numerous illustrations are devoted to the microscopic appearance of some fungous cultures, with many fine details (for instance, the length of primary and secondary conidiophores) which could easily be omitted in a book about the "elements" of medical mycology. On the contrary, the gross morphology of many cultures is described so briefly that a beginner in this field will hardly be able to realize how they look. Nothing is said about the gross appearance of Candida albicans and Coccidioides immitis on Sabouraud's medium. Even a detailed description of cultures is insufficient unless it is supported by pictures. In this respect the book is deficient. Over forty species of fungi are described, and one finds only one picture of a fungous colony, namely, Hormodendrum pedrosoi, which is a rarity and of not much practical importance. In a book on medical mycology, whether elementary or not, all cultures described in the text should be illustrated by pictures, at least in black and white and preferably in natural colors.

The author recognizes that his desire to be brief forced him to sacrifice many details and caused a somewhat dogmatic exposition of the subject. This is noticeable in many parts of the book. For instance, in the discussion of relation of fungi to disease, onychomycosis is noted only in connection with Trichophyton schoenleinii, Trichophyton rubrum and Trichophyton rosaceum, while in reality the nails may be affected by other species of this genus mentioned in that chapter, namely, gypseum, violaceum, crateriforme and acuminatum. On page 52 the author says that "erosio interdigitalis blastomycetica is an intertrigo affecting the web between the third and fourth fingers of the hand." By adding only one word, "mostly," this statement can be made less dogmatic and more nearly correct. The possibility of cure without roentgen epilation in cases of tinea capitis due to Microsporon lanosum is not mentioned. On page 71 the author states that this species is the most frequently encountered on the Atlantic seaboard. This statement may be true for some localities but not for the city of New York, where in former years both Microsporon audouini and Microsporon lanosum were equally common; in the present epidemic M. audouini is the prevailing fungus. On page 98 it is stated that in cases of onychomycosis due to fungi trichophytic inflammation about the nail (paronychia) is present. In the reviewer's opinion paronychia is usually present when the infection is due to Monilia albicans but not when it is due to Trichophyton or Epidermophyton. On page 36 the author states that transfer from a fungous colony is successful only when the latter is small. In fact, a transfer will be successful, regardless of the size of the colony, provided it is still alive and not contaminated.

The "Elements" is a practical and useful book on medical mycology. Its value for a dermatologist and a general practitioner could be still more increased by some additions to the text and additional illustrations.

INDEX TO VOLUME 49

The asterisk (*) preceding the page number indicates an original article in the Archives. Subject entries are made for all articles. Author entries are made for original articles and society transactions. Book Reviews, Editorials and Society Transactions are indexed under these headings in their alphabetical order under the letters B, E and S, respectively.

Abnormalities and Deformities: See under names of diseases, organs and regions, as Atrophy; Ectodermal Defect; Nails, abnormalities; Pigmentation; Skin, abnormalities; etc.

Abrasive, clinical observations on use of soaps containing abrasive, *188

Abscess: See also under names of organs and regions, as Lymph Nodes; etc.
  Salmonella cholerae suis as cause of indolent submalities; Sweat Glands; etc.
  cutaneous abscess, 359

Acanthosis nigricans, 378
  nigricans; clinical note on 2 cases, 361
  nigricans (juvenile type), 299

Achromia: See Vitiligo

Achromotrichia: See Hair, color of

Acid, Ascorbic: See Ascorbic Acid
  Cevitamic: See Ascorbic Acid
  Cholic: See under Bile
  Nicotinic: See Nicotinic Acid
  Orcein-Giemsa Stain: See Stains and Staining
  Paraaminobenzoic: See Vitiligo

Acne, Chloracne: See Chlorodiphenyl: Chloronaphthalene
  lessened sensitivity to tuberculin in, *174
  occupational, 443
  rosacea-like tuberculid of Lewandowsky, 296, 453
  use of soaps containing abrasive; clinical observations, *188
  vulgaris; papulonecrotic tuberculid in Negro; its clinical resemblance to acne vulgaris and early syphilis, *351

Acrodermatitis chronica atrophicans, 216
  chronica atrophicans or early scleroderma, 153
  continua (Hallopeau); effect of treatment with sulfapyridine, *273

Acrosclerosis: See Scleroderma

Actinodermatitis: See under Light

Actinomyces, effect of actinomycin, clavacin and tyrothricin on staphylococcal toxin, 62

Actinomycin: See Actinomyces

Actinomycosis, 143, 445
  treated with sulfadiazine, 359

Adrenalectomy: See Adrenals

Adrenals, mycosis fungoides with bullous lesions; special tests and laboratory data indicating adrenal insufficiency, *315
  oxygen consumption of skin and hair growth after adrenalectomy in white rat, 199

Albinism: See Vitiligo

Alden, H. S.: Asbestos corn, *312

Alibour's Water: See Skin, diseases

Alkaloids, dermatitis from, *181

Allergy: See Anaphylaxis and Allergy

Aloe vera plant, *46

Alopecia: See also Hair, removal
  cicatrisata; pseudopelade, 292
  lymphoblastoma; case for diagnosis, 457

Ambler, J. V.: Experience of dermatologist in Southern Pacific, 224

American Board of Dermatology and Syphilology, *133

Amersbach, J. C.: Localized myxedema in association with hyperthyroidism, *415

Amyloidosis cutis or lichen simplex chronicus, 219
  cutis, poikiloderma or lupus erythematosus, 209
  epicellular and pericellular depositions of amyloid as starting point of, 199
  multiple disseminated sphaceloderma with generalized amyloidosis, 76
  of skin, 143

Anacardiaceae: See Plants

Anaphylaxis and Allergy: See also Food, allergy; Hay Fever; etc.
  contact reactions in atopy; contact reactions in various atopic illnesses, 359
  cutaneous autosensitization; role of staphylococci in chronic eczema of hands, *124
  disseminated lupus erythematosus; allergic disease, 280
  histamine-protein complexes; synthesis and immunologic investigation; histamine-azoprotein, 443
  studies in sensitization to skin; production of antibodies to skin by means of synergistic action of homologous skin antigen and staphylococcus toxin, 200

Anderson, C. R.: Bullous lichen sclerosus et atrophicus; its relation to bullous scleroderma, *423

Anderson, N. P.: Coccidioidomycosis (generalized), 149
  Dermatitis actinica, 295
  Epidermodysplasia verruciformis, 83
  Erythema multiforme, 294
  Erythromelalgia and polycythemia, 293
  Generalized dermatophytosis (probably due to Trichophyton purpureum), 146
  Keratosis pilaris of face, 81
  Lichen planus of gums, 84
  Lichen planus with vesicular lesions on buccal mucosa, 148
  Lupus erythematosus with involvement of conjunctiva, 82
  Neutralization as therapeutic principle in contact dermatitis, *176
  Systemic xanthoma, 149

Anemia, hypochromic, with koilonychia (spoon nails), 282
  sickle cell, 446

Aneurysm, syphilitic, of celiac artery, 280

Angina, Vincent's: See Fusospirochetosis

Angiokeratoma, 65
  Fordyce, 202

Angioma: angiomatous leiomyoma, 202
  cavernosum with fibrosis, 68

Anhidrosis: See Sweat Glands

Ankle: See also Foot
  symmetric syphilitic granuloma of elbows or Bowen's disease of ankle, 289

Antigens and Antibodies: See Lipoids; and under specific antigens and reactions, as Eczema; etc.

Anus, epithelioma of, 450

Aphthae: See Stomatitis

Apparatus: See also Instruments
  calibration of x-ray equipment for superficial therapy, *250

Applestein, R.: Parapsoriasis guttata; case for diagnosis, 203

Armed Forces Personnel: See also Military Medicine
  dermatologists of United States and Canada in active service in present war, 437

Armies: See Military Medicine; War

Arnold, H. L., Jr.: Atypical lupus erythematosus or Senear-Usher syndrome, 297
  Erythema nodosum or periarteritis nodosa, 298
  Lichen sclerosus et atrophicus (Hallopeau), 299
  Neural leprosy with mal perforans, 297

INDEX TO VOLUME 49

The asterisk (*) preceding the page number indicates an original article in the Archives. Subject entries are made for all articles. Author entries are made for original articles and society transactions. Book Reviews, Editorials and Society Transactions are indexed under these headings in their alphabetical order under the letters B, E and S, respectively.

Abnormalities and Deformities: See under names of diseases, organs and regions, as Atrophy; Ectodermal Defect; Nails, abnormalities; Pigmentation; Skin, abnormalities; etc.

Abrasive, clinical observations on use of soaps containing abrasive, *188

Abscess: See also under names of organs and regions, as Lymph Nodes; etc.
  Salmonella cholerae suis as cause of indolent submalities; Sweat Glands; etc.
  cutaneous abscess, 359

Acanthosis nigricans, 378
  nigricans; clinical note on 2 cases, 361
  nigricans (juvenile type), 299

Achromia: See Vitiligo

Achromotrichia: See Hair, color of

Acid, Ascorbic: See Ascorbic Acid
  Cevitamic: See Ascorbic Acid
  Cholic: See under Bile
  Nicotinic: See Nicotinic Acid
  Orcein-Giemsa Stain: See Stains and Staining
  Paraaminobenzoic: See Vitiligo

Acne, Chloracne: See Chlorodiphenyl; Chloronaphthalene
  lessened sensitivity to tuberculin in, *174
  occupational, 443
  rosacea-like tuberculid of Lewandowsky, 296, 453
  use of soaps containing abrasive; clinical observations, *188
  vulgaris; papulonecrotic tuberculid in Negro; its clinical resemblance to acne vulgaris and early syphilis, *351

Acrodermatitis chronica atrophicans, 216
  chronica atrophicans or early scleroderma, 153
  continua (Hallopeau); effect of treatment with sulfapyridine, *273

Acrosclerosis: See Scleroderma

Actinodermatitis: See under Light

Actinomyces, effect of actinomycin, clavacin and tyrothricin on staphylococcal toxin, 62

Actinomycin: See Actinomyces

Actinomycosis, 143, 445
  treated with sulfadiazine, 359

Adrenalectomy: See Adrenals

Adrenals, mycosis fungoides with bullous lesions; special tests and laboratory data indicating adrenal insufficiency, *315
  oxygen consumption of skin and hair growth after adrenalectomy in white rat, 199

Albinism: See Vitiligo

Alden, H. S.: Asbestos corn, *312

Alibour's Water: See Skin, diseases

Alkaloids, dermatitis from, *181

Allergy: See Anaphylaxis and Allergy

Aloe vera plant, *46

Alopecia: See also Hair, removal
  cicatrisata; pseudopelade, 292
  lymphoblastoma; case for diagnosis, 457

Ambler, J. V.: Experience of dermatologist in Southern Pacific, 224

American Board of Dermatology and Syphilology, *133

Amersbach, J. C.: Localized myxedema in association with hyperthyroidism, *415

Amyloidosis cutis or lichen simplex chronicus, 219
  cutis, poikiloderma or lupus erythematosus, 209
  epicellular and pericellular depositions of amyloid as starting point of, 199
  multiple disseminated sphaceloderma with generalized amyloidosis, 76
  of skin, 143

Anacardiaceae: See Plants

Anaphylaxis and Allergy: See also Food, allergy; Hay Fever; etc.
  contact reactions in atopy; contact reactions in various atopic illnesses, 359
  cutaneous autosensitization; role of staphylococci in chronic eczema of hands, *124
  disseminated lupus erythematosus; allergic disease, 280
  histamine-protein complexes; synthesis and immunologic investigation; histamine-azoprotein, 443
  studies in sensitization to skin; production of antibodies to skin by means of synergistic action of homologous skin antigen and staphylococcus toxin, 200

Anderson, C. R.: Bullous lichen sclerosus et atrophicus; its relation to bullous scleroderma, *423

Anderson, N. P.: Coccidioidomycosis (generalized), 149
  Dermatitis actinica, 295
  Epidermodysplasia verruciformis, 83
  Erythema multiforme, 294
  Erythromelalgia and polycythemia, 293
  Generalized dermatophytosis (probably due to Trichophyton purpureum), 146
  Keratosis pilaris of face, 81
  Lichen planus of gums, 84
  Lichen planus with vesicular lesions on buccal mucosa, 148
  Lupus erythematosus with involvement of conjunctiva, 82
  Neutralization as therapeutic principle in contact dermatitis, *176
  Systemic xanthoma, 149

Anemia, hypochromic, with koilonychia (spoon nails), 282
  sickle cell, 446

Aneurysm, syphilitic, of celiac artery, 280

Angina, Vincent's: See Fusospirochetosis

Angiokeratoma, 65
  Fordyce, 202

Angioma; angiomatous leiomyoma, 202
  cavernosum with fibrosis, 68

Anhidrosis: See Sweat Glands

Ankle: See also Foot
  symmetric syphilitic granuloma of elbows or Bowen's disease of ankle, 289

Antigens and Antibodies: See Lipoids; and under specific antigens and reactions, as Eczema; etc.

Anus, epithelioma of, 450

Aphthae: See Stomatitis

Apparatus: See also Instruments
  calibration of x-ray equipment for superficial therapy, *250

Applestein, R.: Parapsoriasis guttata; case for diagnosis, 203

Armed Forces Personnel: See also Military Medicine
  dermatologists of United States and Canada in active service in present war, 437

Armies: See Military Medicine; War

Arnold, H. L., Jr.: Atypical lupus erythematosus or Senear-Usher syndrome, 297
  Erythema nodosum or periarteritis nodosa, 298
  Lichen sclerosus et atrophicus (Hallopeau), 299
  Neural leprosy with mal perforans, 297

Arnold, H. L., Jr.—Continued
Pemphigus erythematosus (Senear-Usher), 298
Pityriasis rubra pilaris, 301
Tuberculoid leprosy; case for diagnosis, 299

Arsenic and Arsenic Compounds: See also Arsphenamines; Tryparsamide
arsenoresistant syphilis (improved), 150
arsenoresistant syphilis, pyogenic ulceration and destruction of nose, 77
concentration of arsenic in blood after administration of mapharsen by rapid drip method; clinical and experimental studies, *321
dermatitis, *180
hyperpigmentation (arsenic?), 70
primary syphilis treated by 26 week course of mapharsen and bismuth; acute basilar meningitis with neuroretinitis developing during treatment, *109
relation of blood ascorbic acid clearance to arsenical intolerance in syphilotherapy, 444
Therapy: See under Lupus erythematosus; Syphilis
toxicity and therapeutic effectiveness of mapharsen by intramuscular administration, 62
unusually high icterus index in patient with fatal hepatic necrosis following mapharsen, 359

Arsphenamines, dermatitis exfoliativa (due to arsphenamine) with hyperkeratosis of palms and soles, 145
misuse of intravenous N. A. B. [neoarsphenamine] for Vincent's infection, 283
postarsphenamine lichen planus, 61
Therapy: See under Syphilis

Arteries: See Aneurysm; Blood pressure; Embolism; Periarteritis; etc.

Arthritis, arthritic symptoms recurring after erysipeloid, 368
gold dermatitis with spectrographic evidence of deposition of gold in skin, 365
keratosis blennorrhagica without gonorrhea (Reiter's disease?), *389

Asbestos corn, *312

Ascorbic Acid, relation of blood ascorbic acid clearance to arsenical intolerance in syphilotherapy, 444

Aspergillus, effect of actinomycin, clavacin and tyrothricin on staphylococcal toxin, 62

Asthma: See Anaphylaxis and Allergy

Astrachan, G. D.: Arsenoresistant syphilis (improved), 150
Arsenoresistant syphilis, pyogenic ulceration and destruction of nose, 77

Athlete's Foot: See Ringworm

Atopy: See Anaphylaxis and Allergy

Atrophoderma: See Atrophy

Atrophy: See also Acrodermatitis chronica atrophicans; and under names of organs and regions
early stage of idiopathic atrophy; case for diagnosis, 81
honeycomb, 444

Australia, calibration of x-ray equipment for superficial therapy, *250
colloid degeneration of skin; report of 3 cases from Australia, *234
contact dermatitis due to capeweed, *331

Avitaminoses: See under Vitamins

Axilla: See Sweat Glands

Ayres, S., Jr.: Chronic benign pemphigus; case for diagnosis, 458
Cutis hyperelastica (Ehlers-Danlos syndrome), 150
Dermatitis herpetiformis limited to scalp, 453
Epitheliomas (self healing), 147
Epitheliomas with spontaneous involution, 83
Generalized dermatophytosis (probably due to Trichophyton purpureum), 146
Hodgkin's disease with cutaneous manifestations, 457
Hydroa estivale with secondary cutaneous changes, 84
Lupus erythematosus complicated by dermatitis factitia, 456
Lupus erythematosus disseminatus, 294
Lymphoblastoma; case for diagnosis, 457
Multiple ulcerations of legs, 455
Sporotrichosis, 454
Xanthoma diabeticorum; case for diagnosis, 292

BCG: See under Tuberculosis

Bacteria: See also Staphylococci; Streptococci; Viruses; etc.
Cholerae: See under Salmonella
erysipelothrix; Erysipelothrix rhusiopathiae septicemia, 139
erysipelothrix; sulfonamide compounds in treatment of Erysipelothrix rhusiopathiae infections; effectiveness of sulfanilamide, sulfapyridine, sulfathiazole and sulfadiazine against experimental infection in mouse and against erysipeloid of Rosenbach in man, *27
Leprosy: See Leprosy
pyocyaneus; greenish pigmentation of nail plates from Bacillus pyocyaneus infection; report of 2 cases, *136

Baer, R. L.: Lupus erythematosus disseminatus (treated with mapharsen), 151

Balanitis: See Penis

Balanosclerosis: See Penis

Baldness: See Alopecia

Bancroft, I.: Contact dermatitis due to hair lacquer, 454
Multiple idiopathic hemorrhagic sarcoma (Kaposi), 295

Barium, dermatitis from, *180

Barritt, M. M.: Rapid macroflocculation test for syphilis; method of Rappaport and Eichhorn, *240

Bazin's Disease: See Erythema induratum

Bean, W. B.: Asymmetric cutaneous lesions in pellagra, *335

Beerman, H.: Case for diagnosis, 381
Chronic lymphatic leukemia, complicated by vaccination, 288
Mycosis fungoides (relationship to congenital ichthyosiform erythroderma), 366

Behcet's Syndrome: See Genitals, ulcers; Stomatitis, aphthous

Beinhauer, L. G.: Clinical effects of paraaminobenzoic acid in vitiligo, *132

Bell's Palsy: See Paralysis, facial

Bella Gutti: See under Plants

Benedek, T.: Epidemiology of tinea capitis; study of tinea capitis in dispensary, *120

Bentonite, *264

Berberian, D. A.: Cutaneous leishmaniasis (Oriental sore); time required for development of immunity after vaccination, *433

Berlin, C.: Behcet's syndrome with involvement of central nervous system; report of case, with necropsy, of lesions of mouth, genitalia and eyes; review of literature, *227

Bernstein, E. T.: Multiple disseminated sphaceloderma with generalized amyloidosis, 76
Mycosis fungoides simulating parapsoriasis, 74
Mycosis fungoides (tumor stage), 74

Besnier-Boeck's Disease: See Sarcoidosis

Bhilawanol: See under Plants

Bilirubin in Blood: See Blood, bilirubin

Binford, C. H.: Chromoblastomycosis; report of case from continental United States and discussion of classification of causative fungus, *398

Bismuth and Bismuth Compounds, primary syphilis treated by 26 week course of mapharsen and bismuth; acute basilar meningitis with neuroretinitis developing during treatment, *109
selective action of thiobismol on induced malaria, 138
Therapy: See under names of various diseases, i. e., Syphilis; etc.

Blastomycosis: See also Coccidioidomycosis
chromoblastomycosis; report of case from continental United States and discussion of classification of causative fungus, *398
chromomycosis, 199
cured by sulfapyridine and sulfathiazole, 444
cutaneous and systemic, 203
frambesiform syphilid (iododerma, bromoderma and blastomycosis not ruled out), 290

Blennorrhagia: See Gonorrhea; Keratosis blennorrhagica
Blood, bilirubin; unusually high icterus index in patient with fatal hepatic necrosis following mapharsen, 359
concentration of arsenic in blood after administration of mapharsen by rapid drip method; clinical and experimental studies, *321
Diseases: See Anemia; Leukemia; etc.
potassium; potassium chloride therapy and serum potassium in infantile eczema, 140
pressure, low; postural hypotension in tabes dorsalis, 358
proteins; hyperglobulinemia, 280
proteins in disseminated lupus erythematosus, 441
relation of blood ascorbic acid clearance to arsenical intolerance in syphilotherapy, 444
vitamin A and cutaneous diseases, *103
Bloom, D.: Erythema nodosum due to sulfathiazole? 380
Erythema nodosum following sulfathiazole therapy, 155
Misuse of sulfonamide compounds, *249
Bluefarb, S. M.: Association of lupus erythematosus and thyrotoxicosis in brother and sister, *111
Melanoma, 220
Nevus striatus symmetricus unguis; report of case with involvement of thumbs and little fingers, *190
Boeck Sarcoid: See Sarcoidosis
Boil, Jericho: See Leishmaniasis
Bones: See also under names of bones
hereditary ectodermal dysplasia of anhidrotic type, presenting conic tapering of hands, various osseous anomalies and atopic eczema, 371
Book Reviews:
Biology of Acarus Scabiei; R. Friedman, 86
Clinical Laboratory Methods and Diagnosis; R. B. H. Gradwohl, 156
Elements of Medical Mycology; J. H. Swartz, 460
Introduction to Medical Mycology; G. M. Lewis and M. E. Hopper, 225
Lepra: Diagnóstico, formas clínicas, tratamiento; G. Basombrio, 388
Modern Treatment of Syphilis; J. E. Moore, with collaboration of J. E. Kemp and others, 225
Notable Contributors to Knowledge of Syphilis; H. Goodman, 86
Practical Survey of Chemistry and Metabolism of Skin; M. Markowitz, 388
Synopsis of Clinical Syphilis; J. K. Howles, 156
Bordet-Wassermann Reaction: See Syphilis
Bowen, S. S.: Treatment of erysipeloid by cryotherapy, *348
Bowen's Disease: See Cancer, precancer
Brain: See Nervous System; etc.
Syphilis: See Neurosyphilis
Breast, cancer en cuirasse, 444
rodent ulcer following radiodermatitis, 205
Brocq's Disease: See Alopecia cicatrisata
Bromide and Bromine, frambesiform syphilid (iododerma, bromoderma and blastomycosis not ruled out), 290
Bubo, Climatic: See Lymphogranuloma Venereum
Bubonulus: See Lymphogranuloma Venereum
Bullosis: See Vesication
Burkhart, R. J.: Dermatologic significance of tissue eosinophilia, *19
Burky, E. L.: Cutaneous autosensitization; role of staphylococci in chronic eczema of hands, *124
Burns, newer concepts in treatment with suggestions for management of wartime thermal injuries, 444
reduced temperature treatments for burns and frostbite, 63
Buschke's Disease: See Scleroderma
Butterworth, T.: Granuloma annulare; case for diagnosis, 203
Cable Rash: See Industry
Calcification and ossification of steatomas of scrotum; report of case, *12; correction, 304
universal calcinosis in infant, 64
Calcinosis: See Calcification
Calcium and Calcium Compounds, calcium pantothenate for human achromotrichia; lack of value on prolonged administration, 139
Callaway, J. L.: Effectiveness in vitro of sulfonamide compounds on Sporotrichum schenckl; report of 5 cases of sporotrichosis in North Carolina, *305
Callosities: See Keratosis
Cancer: See also Epithelioma; Sarcoma; Tumors; and under names of organs and regions
co-existence of chronic lymphogranuloma and cancer, 201
en cuirasse, 444
precancer; symmetric syphilitic granuloma of elbows or Bowen's disease of ankle, 289
x-ray treatment of malignant disease of skin, 360
Canning and Canned Foods, dermatitis from carrots, *266
Cannon, A. B.: Actinomycosis, 143
Dermatitis exfoliativa (due to arsphenamine) with hyperkeratosis of palms and soles, 145
Gangrene of fingers of unknown cause, 146
Leukoplakia; results of treatment by electrodesiccation, 142
Ochronosis, 66
Perifolliculitis capitis abscedens et suffodiens (due to use of "Teek," hair straightener), 67
Primary and secondary syphilis; chronic lymphatic leukemia, 145
Capeweed, contact dermatitis due to, *331
Carate: See Pinta
Carcinoma: See Cancer
Cardiovascular Diseases, cardiovascular involvement in congenital syphilis, 61
Carrion's Disease: See Oroya Fever
Carrots, dermatitis from, *266
Cells: See also Tissue; etc.
effect of roentgen rays on cell-virus associations, 281
epicellular and pericellular depositions of amyloid as starting point of amyloidosis, 199
Cerebrospinal Fever: See Meningitis
Cevitamic Acid: See Ascorbic Acid
Chancre, chancriform lesions of penis, 80, 148
Soft: See Chancroid
Chancroid, sulfadiazine and sulfathiazole therapy in lymphogranuloma venereum and chancroid, 358
Cheek, epithelioma developing 15 years after tricho x-ray treatment for hair on face, 443
paraffinoma, 216
pigmented lesion of, 65
Chemotherapy: See Chancroid; Neurosyphilis; Syphilis
Chickenpox, keloids as sequel to varicella, *278
Children, epidemic characterized by diversity of lesions in skin and mucous membranes, probably caused by Streptococcus pyogenes, 360
Chlorodiphenyl, outbreak of Halowax acne ("cable rash") among electricians, 138
Chloronaphthalene, outbreak of Halowax acne ("cable rash") among electricians, 138
Chlorophyll ointment in treatment of dermatophytosis of feet and similar lesions, *128
Choked Disk: See Neuritis, optic
Cholesterol: See also Lipoids
mycosis fungoides or extracellular cholesterosis, 148
Christian-Weber's Disease: See Panniculitis
Chromium and Chromium Compounds, dermatitis due to chromates and dichromates, *177
Chromomycosis: See Blastomycosis
Chrysarobin, studies on ointments containing chrysarobin, *1

Chung-Hoon, E. K.: Quiescent tuberculoid leprosy, 300

Cicatrix: See also Pemphigus
treatment of slow granulating sores and hypertrophic scars with zinc iontophoresis, 140

Cinnabar: See Mercury

Cipollaro, A. C.: Chronic radiodermatitis resulting from treatment of port wine mark with radium, 153
Erythema induratum, 385

Civatte's Disease: See Poikiloderma

Clavacin: See Aspergillus

Climacteric: See Menopause

Clothing, delousing by fumigation, 282
dermatitis due to resin-finished shorts and fabrics; investigation of cause, pathogenesis and related phenomena observed in 10 cases, 359
dhobie mark dermatitis, 200

Coal tar in dermatologic preparations, *421
Therapy: See Ringworm

Coccidioidomycosis after immunotherapy, 149
generalized, 149

Cohen, D.: Generalized progressive scleroderma, 215
Local arterial embolism, 219
Pityriasis lichenoides chronica; case for diagnosis, 284

Cold: See also Temperature
hypothermia in experimental infections; effect of hypothermia on tuberculin sensitivity in guinea pigs, 62
reduced temperature treatments for burns and frostbite, 63

Cole, H. N.: Chick embryo antigen test for lymphogranuloma venereum, 357

Colloid Degeneration: See under Skin

Combes, F. C.: Report on case of poikiloderma (Civatte?), 146
Xanthoma tuberosum multiplex, 144

Communicable Diseases: See Meningitis; Syphilis; etc.

Complement Fixation: See under Syphilis

Congress: See Societies

Conjunctiva, lupus erythematosus involving eyelids and conjunctiva, 449
lupus erythematosus with involvement of, 82
ocular pemphigus, 362
pemphigus conjunctivae with scarring of skin; report of 3 additional cases, *113

Conjunctivitis: See also Keratoconjunctivitis
keratosis blennorrhagica without gonorrhea (Reiter's disease?), *389
sap dermatitis and conjunctivitis caused by wild fig (Ficus tumila), 201
severe erythema multiforme; report of 2 cases of type ectodermosis erosiva pluriorificialis, with development of cicatricial conjunctivitis and keratitis in 1 case, *47

Cornbleet, T.: Blood vitamin A and cutaneous diseases, *103
Hidradenitis suppurativa, 285
Sarcoidosis, 213

Cornea, Inflammation: See Keratitis
ocular criteria of deficiency of riboflavin, 283

Cornell, V. A.: Leukemia cutis, 75

Corns, asbestos corn, *312

Corson, E. F.: Angiokeratoma (Fordyce), 202
Epidermolysis bullosa with lesions of mucous membranes, 382
Lupus erythematosus (initiated by sulfanilamide?), 202

Cosmetics, bentonite, *264
contact dermatitis due to hair lacquer, 454
contact dermatitis due to "leg makeup," *197
contact dermatitis from cold permanent waving, *432
contact eczema due to nail polish, *183
dermatitis due to hair lacquer, *136
hair lacquer pad dermatitis, *198
new protective cream, *436
unusual example of dermatitis due to nail polish, *197

Costa, O. G.: American (mucocutaneous) leishmaniasis; observations, *194
Tropical ulcer, *260

Costello, M. J.: Dermatitis venenata due to shoe leather dye, 155
Fungous infection; case for diagnosis, 386
Perifolliculitis abscedens et suffodiens, 154
Tinea corporis with fluorescent lanugo hairs, 385

Cottonseed, cutaneous absorption; value of petrolatum, anhydrous wool fat and other bases in percutaneous absorption of topically applied cottonseed allergen, *427

Counter, C. E.: Lupus erythematosus; case for diagnosis, 80
Pemphigus erythematosus (Senear-Usher), 455
Poikiloderma atrophicans vasculare, 85
Urticaria pigmentosa, 81

Couperus, M.: Kraurosis vulvae, 79
Lupus erythematosus, 82
Pemphigus erythematosus; case for diagnosis, 82

Cowan, A.: Ocular pemphigus, 362

Crawford, G. M.: Keratosis blennorrhagica without gonorrhea (Reiter's disease?), *389

Creams: See Cosmetics

Cryotherapy: See Erysipeloid

Cryptostemma Calendulacea: See Capeweed

Cummer, C. L.: Keratosis palmaris et plantaris, 448
Lichen sclerosus et atrophicus, 449
Neurofibromatosis (3 cases presented from Department of Dermatology and Syphilology, St. Vincent Charity Hospital), 447
Sarcoidosis, 446
Sickle cell anemia, 446

Cutis Hyperelastica: See Skin, abnormalities

Cyanide dermatitis, *176

Cysts: See under names of organs and regions

Dalibour's Water: See Skin, diseases

Danlos-Ehlers Syndrome: See Joints; Skin, abnormalities; Tumors

Davidson, D. M.: Parapsoriasis en plaques or mycosis fungoides, 211

Day, J. K.: Chlorophyll ointment in treatment of dermatophytosis of feet and similar lesions, *128

Deficiency Diseases: See Pellagra; Vitamins; etc.

Degeneration, Amyloid: See Amyloidosis
colloid, of skin (colloid milium), 454
colloid, of skin; report of 3 cases from Australia, *234

Dementia Paralytica, therapeutic malaria for patient who had undergone splenectomy, *137

Dentition: See Teeth

De Oreo, G. A.: Primary syphilis treated by 26 week course of mapharsen and bismuth; acute basilar meningitis with neuroretinitis developing during treatment, *109

Depigmentation: See Pigmentation

Depilation: See Hair, removal

Dermatitis: See also Acrodermatitis; Eczema; Neurodermatitis; etc.
Acneform: See Acne
Actinica: See under Light
Arsphenamine: See Arsphenamines
artefacta, 71
asymmetric cutaneous lesions in pellagra, *335
Atopic: See Eczema; Neurodermatitis
Atrophicans: See Atrophy
Blastomycetic: See Blastomycosis
Contact: See Dermatitis venenata
Dhobie Mark: See under Plants
Eczematous: See Eczema
exfoliativa (due to arsphenamine) with hyperkeratosis of palms and soles, 145
exfoliativa, of undetermined origin, 204
factitia, lupus erythematosus complicated by, 456
factitia or lupus erythematosus, 452
herpetiformis limited to scalp, 453
herpetiformis or benign chronic pemphigus, 362
herpetiformis, pemphigus vulgaris or pemphigus erythematosus, 221

Dermatitis—Continued
Medicamentosa: See under Drugs; and under names of drugs, as Arsenic and Arsenic Compounds; Arsphenamines; Bromide and Bromine; Gold and Gold Compounds; Mercury; Tryparsamide; etc.
Multiformis: See Dermatitis herpetiformis
Occupational: See under Industry
of eyelids, *253
Roentgen Ray: See under Roentgen Rays
sap dermatitis and conjunctivitis caused by wild fig (Ficus tumila), 201
Solar: See under Light
vegetans; case of probable pyodermitis vegetans (Hallopeau) confined to mucous membranes, 372
Venenata: See also Dyes; Plants; Resins; etc.
venenata and keratoconjunctivitis caused by manzanillo tree, *236
venenata; contact dermatitis, 281
venenata; contact dermatitis due to capeweed, *331
venenata; contact dermatitis due to hair lacquer, 454
venenata; contact dermatitis due to "leg makeup," *197
venenata; contact dermatitis from cold permanent waving, *432
venenata; contact-dermatitis-like lesions following intranasal application of ephedrine, 379
venenata due to hair lacquer, *136
venenata due to resin-finished shorts and fabrics; investigation of cause, pathogenesis and related phenomena observed in 10 cases, 359
venenata due to shoes, *191
venenata from carrots, *266
venenata; hair lacquer pad dermatitis, *198
venenata; neutralization as therapeutic principle in contact dermatitis, *176
venenata; unusual example of dermatitis due to nail polish, *197
Dermatologic societies, directory of, 87
Dermatologists of United States and Canada in active service in present war, 437
experience of dermatologist in Southern Pacific, 224
need for more dermatologists in war, 223
Dermatology, American Board of Dermatology and Syphilology, *133
in Army station hospital, *91
Dermatomycosis: See Mycosis, cutaneous
Dermatophytosis: See Mycosis, cutaneous
Dermatoses: See Dermatitis; Eczema; Skin, diseases; etc.
Dhoble Mark Dermatitis: See under Plants
Diabetes Mellitus, granuloma annulare or necrobiosis lipoidica diabeticorum, 284
necrobiosis lipoidica diabeticorum, 206
necrobiosis lipoidica diabeticorum (numerous lesions of lower extremities), 212
necrosis and necrobiosis lipoidica diabeticorum, 370
xanthoma diabeticorum (xanthoma eruptivum), 71
Di Cicco, G.: Coal tar in dermatologic preparations, *421
Diethylstilbestrol: See Estrogens
Diphtheria, faucial and labial diphtheria, 282
ulcerative, of skin; sporadic and epidemic types observed in Haifa, Palestine, *408
Directory of dermatologic societies, 87
Disk, Choked: See Neuritis, optic
Dispensary, epidemiology of tinea capitis; study of tinea capitis in dispensary, *120
Dobes, W. L.: Contact eczema due to nail polish, *183
Dorne, M.: Fungous infection or mycosis fungoides, 218
Granuloma annulare or necrobiosis lipoidica diabeticorum, 284
Dostrovski, A.: Fixed erythema due to sulfanilamide with gradually lessening sensitivity, *418
Downing, J. G.: Coal tar in dermatologic preparations, *421
New protective cream, *436
Drugs, addiction; dermatitis artefacta, 71
Eruptions: See also under names of drugs, as Arsenic and Arsenic Compounds; Arsphenamines; Bromide and Bromine; Gold and Gold Compounds; Mercury; Phenolphthalein and Phenolphthalein Derivatives; Tryparsamide; etc.
eruptions; lupus erythematosus of nose; lichenoid drug eruption or lichen planus; lichen planus of thumb nails, 378
Duhring's Disease: See Dermatitis herpetiformis
Durand-Nicolas-Favre's Disease: See Lymphogranuloma Venereum
Dyes, dermatitis venenata due to shoe leather dye, 155
Dyshidrosis: See under Sweat Glands
Dyskeratosis: See Keratosis
Ebert, M. H.: Acrodermatitis chronica atrophicans, 216
Lupus erythematosus or syphilis, 218
Paraffinoma, 216
Sarcoidosis, 213
Ectodermal Defect, hereditary ectodermal dysplasia of anhidrotic type, presenting conic tapering of hands, various osseous anomalies and atopic eczema, 371
hereditary ectodermal dysplasia; report of case, with experimental study, *410
Ectodermosis Erosiva Pluriorificialis: See Erythema multiforme
Eczema, allergy to food odors; its relation to management of infantile eczema, 62
contact dermatitis due to capeweed, *331
contact, due to nail polish, *183
cutaneous autosensitization; role of staphylococci in chronic eczema of hands, *124
hereditary ectodermal dysplasia of anhidrotic type, presenting conic tapering of hands, various osseous anomalies and atopic eczema, 371
nosocomial transmission of eczema vaccinatum, 64
paratraumatic (pyogenic) eczema, 64
potassium chloride therapy and serum potassium in infantile eczema, 140
reaction-producing antigens in eczema of infancy and childhood, 201
squamous, or psoriasis, 301
Eddy, C. E.: Calibration of x-ray equipment for superficial therapy, *250
Edema: See under names of organs
EDITORIALS:
Campaign against leprosy in Cuba, 223
Need for more dermatologists in war, 223
Ehlers-Danlos Syndrome: See Joints; Skin, abnormalities; Tumors
Eichhorn, rapid macroflocculation test for syphilis; method of Rappaport and Eichhorn, *240
Elbows, symmetric syphilitic granuloma of elbows or Bowen's disease of ankle, 289
Electrocoagulation: See under specific headings
Electrodesiccation: See Tongue
Electron Microscope: See Spirochaeta Pallida
Electrosurgery: See under specific headings
Ellis, F. A.: Contact dermatitis due to "leg makeup," *197
Hair lacquer pad dermatitis, *198
Embolism, local arterial, 219
Emmons, C. W.: Chromoblastomycosis; report of case from continental United States and discussion of classification of causative fungus, *398
Endocrine Glands, endocrine puzzle of heterosexual hypertrichosis (hirsutism), 200
Eosinophilia, dermatologic significance of tissue eosinophilia, *19
Eosinophils, eosinophilic granuloma; case for diagnosis, 375
Ephedrine and Ephedrine Derivatives, contact-dermatitis-like lesions following intranasal application of ephedrine, 379
Epidermodysplasia verruciformis, 83, 217, 365

Epidermolysis bullosa, 285, *333
bullosa or lupus erythematosus, 287
bullosa with lesions of mucous membranes, 382

Epidermomycosis; Epidermophytosis: See Mycosis, cutaneous; Ringworm

Epilation: See Hair, removal

Epithelioma, balanosclerosis with beginning epithelioma, 205
leukoplakia of penis with early epitheliomatous changes, 286
of anus, 450
of cheek developing 15 years after tricho x-ray treatment for hair on face, 443
self healing, 147
tertiary syphilis of skin or multiple epithelioma of skin, 206
with spontaneous involution, 83
x-ray treatment of malignant disease of skin, 360

Epstein, N.: Benign neoplasm of tongue (gumma?), 303

Epstein, S.: Lupus erythematosus involving eyelids and conjunctiva, 449

Eruptions: See under Arsenic and Arsenic Compounds; Arsphenamines; Drugs; Skin, diseases; etc.

Erysipeloid, arthritic symptoms recurring after erysipeloid, 368
Erysipelothrix rhusiopathiae septicemia, 139
sulfonamide compounds in treatment of Erysipelothrix rhusiopathiae infections; effectiveness of sulfanilamide, sulfapyridine, sulfathiazole and sulfadiazine against experimental infection in mouse and against erysipeloid of Rosenbach in man, *27
treatment by cryotherapy, *348

Erysipelothrix: See Bacteria, erysipelothrix

Erythema ab igni (from application of heat), 69
annulare centrifugum, 302
fixed, due to sulfanilamide with gradually lessening sensitivity, *418
induratum, 385
multiforme, 294
multiforme bullosum or localized pemphigus vulgaris, 364
multiforme; case for diagnosis, 71
multiforme; report on case for diagnosis, 146
multiforme, severe; report of 2 cases of type ectodermosis erosiva pluriorificialis, with development of cicatricial conjunctivitis and keratitis in 1 case, *47
nodosum due to sulfathiazole? 380
nodosum following sulfathiazole therapy, 155
nodosum or periarteritis nodosa, 298
palmare, 451
recurring erythematous papulonodular syphilis, 374

Erythrasma: See Microsporosis

Erythroderma, Exfoliativum: See Dermatitis exfoliativa
Ichthyosiform: See Ichthyosis
Maculopapular: See Parapsoriasis

Erythromelalgia and polycythemia, 293

Esthiomene: See Lymphogranuloma Venereum

Estradiol: See Estrogens

Estrogens, bullous lichen planus (?) cured by estradiol benzoate, 290
increased resistance to viral infection as result of increased fluid in tissues, 200
keratoderma climactericum; cures with natural and with synthetic estrogen, *254
lichen chronicus simplex associated with hypoestrogenic keratoderma, 451

Evans, V. J.: Nutritional dermatoses in rat; comparison of disseminated neurodermatitis and experimental magnesium deficiency, *33; correction, 226

Exostosis, subungual, 451

Extremities: See also Legs; etc.
Blood Supply: See also Raynaud's Disease; etc.
blood supply; cutaneous manifestations of circulatory disorders of foot, 442
necrobiosis lipoidica diabeticorum (numerous lesions of lower extremities), 212

Eyelashes, Microsporon audouini infection of, *436

Eyelids, contact eczema due to nail polish, *183
dermatitis of, *253
dermatologic lesions about eyes, 138
lupus erythematosus involving eyelids and conjunctiva, 449

Eyes: See also under names of special structures and diseases of eye
Behcet's syndrome with involvement of central nervous system; report of case, with necropsy, of lesions of mouth, genitalia and eyes; review of literature, *227
dermatologic lesions about eyes, 138
injuries; lupus erythematosus following trauma, 366
molluscum contagiosum of, 138

Face, angioma cavernosum with fibrosis, 68
keratosis pilaris of, 81

Fat: See Lipoids

Favre-Nicolas' Disease: See Lymphogranuloma Venereum

Feet: See Foot

Felsher, I. M.: Epidemiology of tinea capitis; study of tinea capitis in dispensary, *120

Felsher, Z.: Hereditary ectodermal dysplasia; report of case, with experimental study, *410

Fever, Artificial: See Dementia Paralytica
Cerebrospinal: See Meningitis
Rocky Mountain Spotted: See Rocky Mountain Spotted Fever
San Joaquin: See Coccidioidomycosis
Therapeutic: See Syphilis; etc.

Fibroma, massive, of scalp, 138

Ficus tumila; sap dermatitis and conjunctivitis caused by wild fig (Ficus tumila), 201

Fig: See Ficus tumila

Filariasis, tropical lymphangitis and abscesses, 140

Fingerprints, congenital absence of, 373

Fingers and Toes: See also Foot; Hand
angiokeratoma, 65
gangrene of fingers of unknown cause, 146
knuckle pads, 144
Nails: See Nails
neural leprosy with mal perforans, 297
nevus striatus symmetricus unguis; report of case with involvement of thumbs and little fingers, *190
ulceration of toe on nevus flammeus; case for diagnosis, 152

Finnerud, C. W.: Necrobiosis lipoidica diabeticorum (numerous lesions of lower extremities), 212
Scleroderma (extensive unilateral morphea of band type), 214

Fluids, increased resistance to viral infection as result of increased fluid in tissues, 200

Fluorescence, tinea corporis with fluorescent lanugo hairs, 385

Fluorides, burns from, *180

Folliculitis capitis abscedens et suffodiens associated with tinea capitis, 364
decalvans with lichen spinulosus, 151
ulerythematosa reticulata; honeycomb atrophy, 444

Food: See also Nutrition; Vitamins
allergy to food odors; its relation to management of infantile eczema, 62

Foot: See also Ankle; Fingers and Toes; etc.
chlorophyll ointment in treatment of dermatophytosis of feet and similar lesions, *128
cutaneous manifestations of circulatory disorders of, 442
dermatitis due to shoes, *191
fungous infections of, 442
inflammatory disorders of skin of feet, 442
rational pharmaceutic treatment of diseases of skin, 223

Fordyce-Fox Disease: See Fox-Fordyce Disease

Formaldehyde, dermatitis from, *180

Formalin: See Verruca

Foster, P. D.: Kraurosis vulvae, 79
Lupus erythematosus, 82
Pemphigus erythematosus; case for diagnosis, 82

Fourth Venereal Disease: See Lymphogranuloma Venereum

Fox, H.: American Board of Dermatology and Syphilology, *133
Amyloidosis of skin, 143
Greenish pigmentation of nail plates from Bacillus pyocyaneus infection; report of 2 cases, *136
Pityriasis rubra pilaris, 69

Fox-Fordyce Disease, case of, 360
leukoplakia and Fordyce's disease, 368

Frambesia, frambesiform syphilid (iododerma, bromoderma and blastomycosis not ruled out), 290

Frank, L. J.: Amyloidosis cutis, poikiloderma or lupus erythematosus, 209
Generalized progressive scleroderma; case for diagnosis, 210
Herpes gestationis; case for diagnosis, 209

Freeman, C.: Relationship of balanitis xerotica obliterans to lichen sclerosus et atrophicus, *57

Frei Reaction: See Lymphogranuloma Venereum

Friedman, R.: Congenital syphilis in woman with exaggerated saber shins and (recently) active gummatous ulceration, 366
Exfoliative dermatitis of undetermined origin, 204
Folliculitis capitis abscedens et suffodiens associated with tinea capitis, 364
Hereditary ectodermal dysplasia of anhidrotic type, presenting conic tapering of hands, various osseous anomalies and atopic eczema, 371
Neurofibroma (plexiform neuroma), 205
Recurrent actinic (solar) dermatitis, 291
Rodent ulcer of breast following radiodermatitis, 205
Tuberculosis of skin (sarcoidosis?), 206

Frost, K. P.: Mycosis fungoides or extracellular cholesterosis, 148
Pemphigus vulgaris, 295
Turban tumor, 147

Frostbite, lupus erythematosus following, 367
reduced temperature treatments for burns and frostbite, 63

Fumigation, delousing of clothing by, 282

Fungi: See also Actinomycosis; Blastomycosis; Mycosis; Ringworm; Trichophytosis; etc.
diagnostic and registry center for fungous diseases of man, 226
fungous infection; case for diagnosis, 386
fungous infection or mycosis fungoides, 218

Fusospirochetosis, misuse of intravenous N. A. B. [neoarsphenamine] for Vincent's infection, 283

Gangrene of fingers of unknown cause, 146

Garb, J.: Mycosis fungoides with bullous lesions; special tests and laboratory data indicating adrenal insufficiency, *315

Garbe, W.: Keratoderma climactericum; cures with natural and with synthetic estrogen, *254

Garner, V. C.: Morphea (for therapeutic suggestions), 384

Gas Masks: See Masks

General Paralysis: See Dementia Paralytica

Genitals: See also under names of genitals, as Penis; etc.
ulcers; Behcet's syndrome with involvement of central nervous system; report of case, with necropsy, of lesions of mouth, genitalia and eyes; review of literature, *227

Gerlach, M. J.: Psoriasiform eruption (psoriasis, tertiary syphilis or parapsoriasis en plaques), 383

Gilchrist's Disease: See Blastomycosis

Gill, S.: Ulcerative diphtheria of skin; sporadic and epidemic types observed in Haifa, Palestine, *408

Ginsburg, L.: Hair lacquer pad dermatitis, *198

Glossitis: See Tongue

Gold and Gold Compounds, dermatitis with spectrographic evidence of deposition of gold in skin, 365
Therapy: See under Vitiligo; etc.

Goldberg, H. C.: Aloe vera plant, *46
Area factor in roentgen irradiation, *346

Goldberger, L. A.: Multiple disseminated sphacelo-derma with generalized amyloidosis, 76
Mycosis fungoides simulating parapsoriasis, 74
Mycosis fungoides (tumor stage), 74

Goldblatt, S.: Intensive ambulatory therapy of syphilis; 30 day mapharsen technic, *403

Goldman, L.: Greenish pigmentation of nail plates from Bacillus pyocyaneus infection; report of 2 cases, *136

Goldstein, L.: Hemangoendothelioma, 287
Prurigo, 371

Gonorrhea: See also Venereal Diseases
of skin, *436
primary gonorrheal cutaneous infection, 61

Goodman, H.: Bentonite, *264
Rational pharmaceutic treatment of diseases of skin, *16

Gordon Test: See Hodgkin's Disease

Grafts: See under Skin

Graham, J. C.: Angiokeratoma, 65
Xanthoma diabeticorum (xanthoma eruptivum), 71

Graham, T.: Lichen nitidus, 381

Gramacidin; effect of actinomycin, clavacin and tyrothricin on staphylococcal toxin, 62

Granuloma annulare; case for diagnosis, 203
annulare of lip, 289
annulare or necrobiosis lipoidica diabeticorum, 284
eosinophilic; case for diagnosis, 375
Fungoides: See Mycosis fungoides
Inguinale: See Lymphogranuloma Venereum
Malignant: See Hodgkin's Disease
Paracoccidioidal: See Blastomycosis
symmetric syphilitic granuloma of elbows or Bowen's disease of ankle, 289
Venereal: See Lymphogranuloma Venereum

Greenbaum, S. S.: Case for diagnosis, 384
Case of probable pyodermitis vegetans (Hallopeau) confined to mucous membranes, 372
Hemangoendothelioma, 287
Prurigo, 371
Tuberculosis cutis; case for diagnosis, 384

Greenberg, S. I.: Epidermolysis bullosa, *333

Griswold, C. M.: Treatment of erysipeloid by cryotherapy, *348

Gruhzit, O. M.: Concentration of arsenic in blood after administration of mapharsen by rapid drip method; clinical and experimental studies, *321

Guequierre, J. P.: Pemphigus vulgaris (recovered), 383

Gumma: See Syphilis, tertiary

Gums, Diseases: See Fusospirochetosis
lichen planus of, 84

Habermann's Disease: See Pityriasis lichenoides

Hagedorn, replaceable Hagedorn needle with handle as scalpel, *198

Hailey, H. E.: Lupus erythematosus with false positive Kahn reactions, *355

Hair, color of; calcium pantothenate for human achromotrichia; lack of value on prolonged administration, 139
color of; vitamins for gray hair (achromotrichia), 63
Diseases: See Alopecia
excessive; endocrine puzzle of heterosexual hypertrichosis (hirsutism), 200
Lacquer: See Cosmetics
oxygen consumption of skin and hair growth after adrenalectomy in white rat, 199
perifolliculitis capitis abscedens et suffodiens, 67
removal; epithelioma of cheek developing 15 years after tricho x-ray treatment for hair on face, 443
removal; ringworm of scalp; successful use of roentgen rays to epilate local areas of infection, *107
tinea corporis with fluorescent lanugo hairs, 385

Hall, A. F.: Rosacea-like tuberculid of Lewandowsky, 453

Hallopeau's Disease: See Acrodermatitis

Halowax: See Chlorodiphenyl; Chloronaphthalene

Hand, E. A.: Colloid degeneration of skin; report of 3 cases from Australia, *234
Contact dermatitis due to capeweed, *331

Hand: See also Fingers and Toes
cutaneous autosensitization; role of staphylococci in chronic eczema of hands, *124
hereditary ectodermal dysplasia of anhidrotic type, presenting conic tapering of hands, various osseous anomalies and atopic eczema, 371

Hand Cream: See Cosmetics

Hansen's Disease: See Leprosy

Hardy, M. K.: Sarcoidosis, 382

Harley, R. D.: Dermatitis venenata and keratoconjunctivitis caused by manzanillo tree, *236

Hay Fever: See also Anaphylaxis and Allergy
contact dermatitis due to capeweed, *331

Hazen, H. H.: Dermatitis of eyelids, *253

Heat: See also Temperature
erythema ab igni from application of, 69

Hemangioma, cavernous, 369

Hemangioendothelioma, 287

Hemopoietic System, Diseases: See Anemia; Leukemia; etc.

Henningsen, A. B.: Acrodermatitis chronica atrophicans, 216
Epidermolysis bullosa, 285
Keratoma dissipatum palmoplantare (Brauer) (keratoderma papulosum), 217
Pemphigus vulgaris, pemphigus erythematosus or dermatitis herpetiformis, 221

Heredity, xeroderma pigmentosum; report of case and consideration of incomplete sex linkage in inheritance of disease, *157

Herpes gestationis; case for diagnosis, 209
zoster and Bell's palsy, 445
zoster, motor complications of, 139

Hess, G.: Chromoblastomycosis; report of case from continental United States and discussion of classification of causative fungus, *398

Hetreed, F. W.: Sarcoid; case for diagnosis, 284

Hewish, E. A.: Rapid macroflocculation test for syphilis; method of Rappaport and Eichhorn, *240

Hidradenitis: See under Sweat Glands

Hinton Test: See Syphilis

Hippomane Mancinella: See Manzanillo Tree

Hirsutism: See Hair, excessive

Histamine; histamine-protein complexes; synthesis and immunologic investigation; histamine-azoprotein, 443
in Blood: See under Blood

Histiocytosis, nodular; case for diagnosis, 302

Hodgkin's Disease of skin, 289
of skin in Negro, 75
with cutaneous manifestations, 457

Hollander, L.: Replacement graft in surgical treatment of lupus vulgaris, *60

Hopkins, H. H.: Cutaneous autosensitization; role of staphylococci in chronic eczema of hands, *124

Hopper, M. E.: Ringworm of scalp; successful use of roentgen rays to epilate local areas of infection, *107

Hormones: See Endocrine Glands; Estrogens; Pituitary Preparations; etc.
Melanotropic: See Pituitary Preparations

Hospitals, dermatology in Army Station Hospital, *91

Howell, J. B.: Contact dermatitis from cold permanent waving, *432

Howell, W. M.: Asbestos corn, *312

Hydroa estivale with secondary cutaneous changes, 84

Hyperglobulinemia: See Blood, proteins

Hyperkeratosis: See Keratosis

Hyperpigmentation: See Pigmentation

Hyperpyrexia: See under Dementia Paralytica; Syphilis; etc.

Hypertension: See Blood pressure, high

Hyperthyroidism: See under Thyroid

Hypothermia: See Cold

Hypotrichosis: See Alopecia

Ichthyosis, mycosis fungoides (relationship to congenital ichthyosiform erythroderma), 366

Icteric Index: See Blood, bilirubin

Immunity: See Anaphylaxis and Allergy

Impetigo contagiosa, etiology of, 281

Indians, American; relation of blood ascorbic acid clearance to arsenical intolerance in syphilotherapy, 444

Industry, asbestos corn, *312
contact dermatitis, 281
dermatitis from carrots, *266
new protective cream, *436
occupational acne, 443
outbreak of Halowax acne ("cable rash") among electricians, 138

Infants, idiosyncrasy to mercury preparations in childhood; report of 2 cases of reactions to 5 per cent ammoniated mercury ointment and mercury bichloride solution (1:4,000), 280
newborn, congenital defect of skin in, 280

Infectious Diseases: See Diphtheria; Syphilis; etc.

Injections, Intramuscular: See under Arsenic and Arsenic Compounds

Injuries: See Eyes; Nose; etc.

Instruments: See also Apparatus
replaceable Hagedorn needle with handle as scalpel, *198

Intertrigo, pseudomembranous, case of, 360

Iodine and Iodine Compounds, dermatitis due to iodine, *179
frambesiform syphilid (iododerma, bromoderma and blastomycosis not ruled out), 290

Irgang, S.: Papulonecrotic tuberculid in Negro; its clinical resemblance to acne vulgaris and early syphilids, *351

Iris, psoriasis and eye color, 360

Jacobi-Petges' Disease: See Poikiloderma atrophicans vasculare

Jacobson, H. P.: Coccidioidomycosis after immunotherapy, 149

Jensen, A.: Lupus vulgaris, 455
Lymphoblastoma; case for diagnosis, 457

Jericho Boil: See Leishmaniasis

Johnson, H. M.: Acanthosis nigricans (juvenile type), 299
Balanitis xerotica obliterans, 299
Case for diagnosis, 302

Johnson-Stevens Disease: See Erythema multiforme

Joints, cutis hyperelastica (Ehlers-Danlos syndrome), 150

Kahn Test: See also under Syphilis
lupus erythematosus with false positive Kahn reactions, *355
serologic change occurring during short courses of neoarsphenamine and bismuth, such as were possible while patients stayed at venereal diseases center for African soldiers, 361

Kala-Azar: See Leishmaniasis

Kanee, B.: Localized myxedema in association with hyperthyroidism, *415

Kaposi's Sarcoma: See Sarcoma
Varicelliform Eruption: See Pyoderma

Katz, S.: Case for diagnosis, 384
Tuberculosis cutis; case for diagnosis, 384

Keloids: See also Cicatrix
as sequel to varicella, *278
review of literature and report of 80 cases, 441

Keratitis: See also Keratoconjunctivitis
severe erythema multiforme; report of 2 cases of type ectodermosis erosiva pluriorificialis, with development of cicatricial conjunctivitis and keratitis in 1 case, *47

Keratoconjunctivitis and dermatitis venenata caused by manzanillo tree, *236
Keratoderma: See Keratosis
Keratoma: See Keratosis
Keratosis: See also Ichthyosis
biennorrhagica without gonorrhea (Reiter's disease?), *389
congenital absence of fingerprints, 373
dermatitis exfoliativa (due to arsphenamine) with hyperkeratosis of palms and soles, 145
keratoderma biennorrhagica; some further observations as to etiology, 360
keratoderma climactericum; case for diagnosis, 377
keratoderma climactericum; cures with natural and with synthetic estrogen, *254
keratoma dissipatum palmoplantare (Brauer) (keratoderma papulosum), 217
lichen chronicus simplex associated with hypoestrogenic keratoderma, 451
palmaris et plantaris, 448
pilaris of face, 81
Kile, R. L.: Clinical observations on use of soaps containing abrasive, *188
Killian, J.: Effectiveness in vitro of sulfonamide compounds on Sporotrichum schenckl; report of 5 cases of sporotrichosis in North Carolina, *305
Klauder, J. V.: Arthritic symptoms recurring after erysipeloid, 368
Case of probable pyodermitis vegetans (Hallopeau) confined to mucous membranes, 372
Ocular pemphigus, 362
Sulfonamide compounds in treatment of Erysipelothrix rhusiopathiae infections; effectiveness of sulfanilamide, sulfapyridine, sulfathiazole and sulfadiazine against experimental infection in mouse and against erysipeloid of Rosenbach in man, *27
Knee, lupus vulgaris of, 154
Knuckle Pads: See Fingers and Toes
Koilonychia: See Nails
Kraurosis: See Vulva

Labia Majora: See Vulva
Lacquer, contact dermatitis due to hair lacquer, 454
dermatitis due to hair lacquer, *136
hair lacquer pad dermatitis, *198
Nail Lacquer: See Cosmetics
LaRocco, C. G.: Neurofibromatosis (3 cases presented from Department of Dermatology and Syphilology, St. Vincent Charity Hospital), 447
Sarcoidosis, 446
Sickle cell anemia, 446
Lawrence, H.: Case for diagnosis, 300
Lichen nitidus, 301
Psoriasis of nails, 301
Laymon, C. W.: Relationship of balanitis xerotica obliterans to lichen sclerosus et atrophicus, *57
Scleredema adultorum, 452
Leather, dermatitis venenata due to shoe leather dye, 155
Legs: See also Extremities; Foot
contact dermatitis due to "leg makeup," *197
Ulcers: See Ulcers
Leifer, W.: Balanitis xerotica obliterans; report of case, *118
Leiomyoma, angiomatous, 202
Leishmaniasis, American (mucocutaneous) leishmaniasis; observations, *194
cutaneous (Oriental sore); time required for development of immunity after vaccination, *433
Lentz, J. W.: Nevus comedonicus, 367
Symmetric syphilitic granuloma of elbows or Bowen's disease of ankle, 289
Leprosy, antiquity of congenital syphilis (described as leprosy), 441
campaign against leprosy in Cuba, 223
epidemiology and natural history, 140
neural, with mal perforans, 297
tuberculoid; case for diagnosis, 299
tuberculoid, quiescent, 300
Leukemia cutis, 75
lymphatic, chronic, complicated by vaccination, 288
lymphatic, chronic; primary and secondary syphilis, 145
Leukemid: See Leukemia, lymphatic
Leukocytes: See Eosinophils; Leukemia; etc.
Leukoderma: See Vitiligo
Leukopathia: See Vitiligo
Leukoplakia: See Mouth; Penis; Tongue
Lever, W. F.: Acrodermatitis continua (Hallopeau); effect of treatment with sulfapyridine, *273
Keratosis blennorrhagica without gonorrhea (Reiter's disease?), *389
Pemphigus conjunctivae with scarring of skin; report of 3 additional cases, *113
Severe erythema multiforme; report of 2 cases of type ectodermosis erosiva pluriorificialis, with development of cicatricial conjunctivitis and keratitis in 1 case, *47
Levin, O. L.: Hodgkin's disease of skin in Negro, 75
Melanoma; regression with roentgen ray therapy, 78
Lewis, G. M.: Angioma cavernosum fibrosis, 68
Contact-dermatitis-like lesions following intranasal application of ephedrine, 379
Eosinophilic granuloma; case for diagnosis, 375
Erythema multiforme; case for diagnosis, 71
Erythema multiforme; report on case for diagnosis, 146
Keratoderma climactericum; case for diagnosis, 377
Lupus erythematosus; case for diagnosis, 69
Lupus erythematosus of nose; lichenoid drug eruption or lichen planus; lichen planus of thumb nails, 378
Ringworm of scalp; successful use of roentgen rays to epilate local areas of infection, *107
Tinea capitis (Trichophyton violaceum), 67
Verruca plana (resistant to treatment), 65
Lice: See Pediculi
Lichen nitidus, 301, 381
nitidus, generalized, 381
planus, bullous (?) cured by estradiol benzoate, 290
planus; lupus erythematosus of nose; lichenoid drug eruption or lichen planus; lichen planus of thumb nails, 378
planus ocreaformis, 450
planus of gums, 84
planus, postarsphenamine, 61
planus with vesicular lesions on buccal mucosa, 148
sclerosus et atrophicans associated with circumscribed scleroderma, 386
sclerosus et atrophicus, 449
sclerosus et atrophicus, bullous; its relation to bullous scleroderma, *423
sclerosus et atrophicus (Hallopeau), 299
sclerosus et atrophicus, relationship of balanitis xerotica obliterans to, *57
Simplex Chronicus: See Neurodermatitis
spinulosus with folliculitis decalvans, 151
Lichenification: See Neurodermatitis
Light, colloid degeneration of skin; report of 3 cases from Australia, *234
dermatitis actinica, 295
hydroa estivale with secondary cutaneous changes, 84
recurrent actinic (solar) dermatitis, 291
Lindsay, H. C. L.: Chancriform penile lesions, 80, 148
Leukoderma acquisitum centrifugum, 459
Linear lymphatic nevus, 458
Parapsoriasis, 293
Parapsoriasis (Haberman?); case for diagnosis, 79
Vitiligo, treated with gold tribromide orally, 293
Lingua: See Tongue
Lipids: See Lipoids
Lipoids, granuloma annulare or necrobiosis lipoidica diabeticorum, 284
necrobiosis lipoidica diabeticorum, 206

Lipoids—Continued
necrobiosis lipoidica diabeticorum (numerous lesions of lower extremities), 212
necrosis and necrobiosis lipoidica diabeticorum, 370
Lips, chronic lymphedema of, 296
Diphtheria: See Diphtheria
granuloma annulare of, 289
hemangoendothelioma, 287
Lisa, J. R.: Multiple idiopathic hemorrhagic sarcoma of Kaposi in full-blooded Negro, *270
Little, R. P.: Alibour's water, 357
Replaceable Hagedorn needle with handle as scalpel, *198
Livedo reticularis, 450
Liver, effect of prolonged tryparsamide therapy on liver function, 61
syphilis of, 358
unusually high icterus index in patient with fatal hepatic necrosis following mapharsen, 359
Ludy, J. B.: Angiomatous leiomyoma, 202
Congenital absence of fingerprints, 373
Dermatitis herpetiformis or benign chronic pemphigus, 362
Gold dermatitis with spectrographic evidence of deposition of gold in skin, 365
Leukoplakia and Fordyce's disease, 368
Lupus erythematosus following frostbite, 367
Lupus erythematosus following trauma, 366, 367
Tuberculous (?) ulcers, 369
Luetin Test: See under Syphilis
Luotest: See under Syphilis
Lupoid: See Sarcoidosis
Lupus: See also Tuberculosis
erythematosus, 82
erythematosus, acute, or acute thrombocytopenia, 205
erythematosus, amyloidosis cutis or poikilioderma, 209
erythematosus and panniculitis of cheeks, 142
erythematosus; association of lupus erythematosus and thyrotoxicosis in brother and sister, *111
erythematosus, atypical, or Senear-Usher syndrome, 297
erythematosus; case for diagnosis, 69, 80, 381
erythematosus complicated by dermatitis factitia, 456
erythematosus, disseminated; allergic disease? 280
erythematosus, disseminated, plasma proteins in, 441
erythematosus disseminatus, 72, 294
erythematosus disseminatus (treated with mapharsen), 151
erythematosus, extensive discoid type, 72
erythematosus following frostbite, 367
erythematosus following trauma, 366, 367
erythematosus (initiated by sulfanilamide?), 202
erythematosus involving eyelids and conjunctiva, 449
erythematosus of nose; lichenoid drug eruption or lichen planus; lichen planus of thumb nails, 378
erythematosus or dermatitis factitia, 452
erythematosus or epidermolysis bullosa, 287
erythematosus or lichenoid sarcoid, 68
erythematosus or syphilis, 218
erythematosus, subacute disseminate, 454
erythematosus with false positive Kahn reactions, *355
erythematosus with involvement of conjunctiva, 82
vulgaris, 155, 455
vulgaris; cutaneous tuberculosis (sarcoids, tuberculids or lupus vulgaris?), 220
vulgaris of knee, 154
vulgaris, replacement graft in surgical treatment of, *60
Lygranum: See Lymphogranuloma Venereum
Lymph Nodes: See also Leukemia, lymphatic; Lymphosarcoma; etc.
tropical lymphangitis and abscesses, 140
Lymphangitis, Tropical: See Filariasis
Lymphedema: See Lips
Lymphoblastoma, case for diagnosis, 457
Lymphogranuloma, Hodgkin's: See Hodgkin's Disease
Inguinale: See Lymphogranuloma Venereum
Schaumann's: See Sarcoidosis
Lymphogranuloma Venereum, chick embryo antigen test for, 357
co-existence of chronic lymphogranuloma and cancer, 201
hyperglobulinemia, 280
sulfadiazine and sulfathiazole therapy in lymphogranuloma venereum and chancroid, 358
Lymphopathia Venereum: See Lymphogranuloma Venereum
Lymphosarcoma, complicated by vaccination, 288
statistical study and evaluation of treatment, 140
Lynch, F. W.: Lessened sensitivity to tuberculin in acne, *174

McGuire, J. A.: Case for diagnosis, 381
Chronic lymphatic leukemia, complicated by vaccination, 288
Frambesiform syphilid (iododerma, bromoderma and blastomycosis not ruled out), 290
Lymphosarcoma, complicated by vaccination, 288
Macklin, M. T.: Xeroderma pigmentosum; report of case and consideration of incomplete sex linkage in inheritance of disease, *157
Macroflocculation Test: See Syphilis
Madden, J. F.: Unusual example of dermatitis due to nail polish, *197
Magnesium, nutritional dermatoses in rat; comparison of disseminated neurodermatitis and experimental magnesium deficiency, *33; correction, 226
Mal del Pinto: See Pinta
Mal Perforans: See Fingers and Toes
Malaria, hemorrhagic purpura due to toxic effects of quinine salts, 445
induced, selective action of thiobismol on, 138
Therapeutic: See also Dementia Paralytica; Syphilis; etc.
therapeutic, for patient who had undergone splenectomy, *137
Mantoux Reaction: See Tuberculosis
Manzanillo Tree, dermatitis venenata and keratoconjunctivitis caused by manzanillo tree; *236
Mapharsen: See Arsenic and Arsenic Compounds; Lupus erythematosus disseminatus; Syphilis
Marking Nut: See under Plants
Masks, treatment of respirator dermatitis (gas mask), 63
Mason, H. S.: Dermatitis from carrots, *266
Mazzini Test: See under Syphilis
Medicine, Military: See Military Medicine
Melanin: See Melanoma; Pigmentation
Melanoma, 153, 220, 457
regression with roentgen ray therapy, 78
Meleda Disease: See Keratosis
Meningitis, primary syphilis treated by 26 week course of mapharsen and bismuth; acute basilar meningitis with neuroretinitis developing during treatment, *109
Menopause, keratoderma climactericum; case for diagnosis, 377
keratoderma climactericum; cures with natural and with synthetic estrogen, *254
Mercury, generalized mercurial (cinnabar) reaction following tattooing, *172
idiosyncrasy to mercury preparations in childhood; report of 2 cases of reactions to 5 per cent ammoniated mercury ointment and mercury bichloride solution (1:4,000), 280
mercurial dermatitis, *180
urticaria following dental silver filling, 443
Metals: See Gold and Gold Compounds; Mercury; etc.
Mibelli's Disease: See Keratosis
Michelson, H. E.: Circumscribed scleroderma, 451
Lupus erythematosus or dermatitis factitia, 452
Microsporosis; erythrasma, 285
Microsporon audouini infection of eyelashes, *436

Military Medicine: See also Hospitals; etc.
dermatitis venenata and keratoconjunctivitis caused by manzanillo tree, *236
dermatology in Army Station Hospital, *91
dhobie mark dermatitis, 200
epidermolysis bullosa, *333
experience of dermatologist in Southern Pacific, 224
sap dermatitis and conjunctivitis caused by wild fig (Ficus tumila), 201

Milium, Colloid: See under Skin

Miller, H.: Erythema annulare centrifugum, 302

Mitchell, J. H.: Cutaneous tuberculosis (sarcoid, tuberculids or lupus vulgaris?), 220
Erythrasma, 285
Leukoplakia of penis with early epitheliomatous changes, 286
Sarcoid; case for diagnosis, 284

Molds: See Fungi

Moles: See Nevi

Molluscum contagiosum of eye, 138

Montgomery, H.: Dermatologic significance of tissue eosinophilia, *19

Moorman, H. E.: Chlorophyll ointment in treatment of dermatophytosis of feet and similar lesions, *128

Morphea: See Scleroderma

Mouth: See also Gums; Stomatitis; etc.
Behcet's syndrome with involvement of central nervous system; report of case, with necropsy, of lesions of mouth, genitalia and eyes; review of literature, *227
leukoplakia and Fordyce's disease, 368
lichen planus with vesicular lesions on buccal mucosa, 148

Mucous Membrane: See also Lips; Mouth; Nose; etc.
case of probable pyodermitis vegetans (Hallopeau) confined to mucous membranes, 372
epidemic in children characterized by diversity of lesions in skin and mucous membranes, probably caused by Streptococcus pyogenes, 360
epidermolysis bullosa with lesions of, 382

Mycobacterium Leprae: See under Leprosy

Mycosis: See also Actinomycosis; Blastomycosis; Moniliasis; Nails; Ringworm; Tinea; etc.
cutaneous: chlorophyll ointment in treatment of dermatophytosis of feet and similar lesions, *128
cutaneous; fungous infections of foot, 442
cutaneous; generalized dermatophytosis (probably due to Trichophyton purpureum), 146
fungoides, 291, 447
fungoides or extracellular cholesterosis, 148
fungoides or fungous infection, 218
fungoides or parapsoriasis en plaques, 211
fungoides preceded by psoriasis vulgaris of 30 years' duration, 143
fungoides (relationship to congenital ichthyosiform erythroderma), 366
fungoides simulating parapsoriasis, 74
fungoides (tumor stage), 74
fungoides with bullous lesions; special tests and laboratory data indicating adrenal insufficiency, *315

Myiasis, dermal; report of 3 cases, *309

Myocarditis, diffuse gummatous, 199

Myxedema, localized, in association with hyperthyroidism, *415

Nails, abnormalities; pachyonychia congenita, 368
greenish pigmentation of nail plates from Bacillus pyocyaneus infection; report of 2 cases, *136
hypochromic anemia with koilonychia (spoon nails), 282
lupus erythematosus of nose; lichenoid drug eruption or lichen planus; lichen planus of thumb nails, 378
nevus striatus symmetricus unguis; report of case with involvement of thumbs and little fingers, *190
Polish: See Cosmetics
psoriasis of, 301
subungual exostosis, 451

Nares: See Nose

Neck, melanoma, 457

Necrobiosis lipoidica diabeticorum, 206
lipoidica diabeticorum and necrosis, 370
lipoidica diabeticorum (numerous lesions of lower extremities), 212
lipoidica diabeticorum or granuloma annulare, 284

Necrosis: See also under Liver
and necrobiosis lipoidica diabeticorum, 370

Needle, replaceable Hagedorn needle with handle as scalpel, *198

Negroes, Hodgkin's disease of skin in Negro, 75
multiple idiopathic hemorrhagic sarcoma of Kaposi in full-blooded Negro, *270
mycosis fungoides, 447

Neoarsphenamine: See Arsphenamines; and under names of various diseases, as Syphilis; etc.

Neoplasms: See Cancer; Sarcoma; Tumors

Nerves: See also Nervous System; Neuritis; Paralysis
neural leprosy with mal perforans, 297

Nervous System: See also Nerves; etc.
Behcet's syndrome with involvement of central nervous system; report of case, with necropsy, of lesions of mouth, genitalia and eyes; review of literature, *227
Syphilis: See Neurosyphilis

Netherton, E. W.: Mycosis fungoides, 447
Poikiloderma of Civatte, 448

Neuritis, optic; primary syphilis treated by 26 week course of mapharsen and bismuth; acute basilar meningitis with neuroretinitis developing during treatment, *109

Neurodermatitis; lichen chronicus simplex associated with hypoestrogenic keratoderma, 451
lichen simplex chronicus or amyloidosis cutis, 219
nutritional dermatoses in rat; comparison of disseminated neurodermatitis and experimental magnesium deficiency, *33; correction, 226

Neurofibroma (plexiform neuroma), 205

Neurofibromatosis, 3 cases presented from Department of Dermatology and Syphilology, St. Vincent Charity Hospital, 447

Neuroma; neurofibroma (plexiform neuroma), 205

Neuroretinitis: See Neuritis, optic; Retinitis

Neurosyphilis: See also Dementia Paralytica; Tabes Dorsalis
effect of prolonged tryparsamide therapy on liver function, 61
relation of trauma to syphilis of nervous system, 138

Nevi comedonicus, 367
flammeus; chronic radiodermatitis resulting from treatment of port wine mark with radium, 153
flammeus; ulceration of toe on nevus flammeus; case for diagnosis, 152
linear lymphatic, 458
striatus symmetricus unguis; report of case with involvement of thumbs and little fingers, *190
Vascular: See Angioma

Newborn Infants: See Infants, newborn

Nicholas, L.: Cutaneous and systemic blastomycosis, 203
Mycosis fungoides, 291

Nicolas-Favre Disease: See Lymphogranuloma Venereum

Nicotinic Acid: See also Vitamins, B
treatment of pellagra with, 141

Nippert, P. H.: Contact eczema due to nail polish, *183

Nitrotoluene, dermatitis due to trinitrotoluene, *178

Nodes: See Erythema nodosum; etc.

Nomenclature, Allbour's water, 357

Noojin, R. O.: Effectiveness in vitro of sulfonamide compounds on Sporotrichum schencki; report of 5 cases of sporotrichosis in North Carolina, *305

Nose: See also Otorhinolaryngology
arsenoresistant syphilis, pyogenic ulceration and destruction of nose, 77
contact-dermatitis-like lesions following intranasal application of ephedrine, 379

Nose—Continued
epitheliomas (self healing), 147
epitheliomas with spontaneous involution, 83
injuries; lupus erythematosus following trauma, 366, 367
lupus erythematosus of; lichenoid drug eruption or lichen planus; lichen planus of thumb nails, 378

Novy, F. G., Jr.: Generalized mercurial (cinnabar) reaction following tattooing, *172
Sarcoid, 302

Nutrition: See also Food; Vitamins; etc.
nutritional dermatoses in rat; comparison of disseminated neurodermatitis and experimental magnesium deficiency, *33; correction, 226

Obermayer, M. E.: Chronic lymphedema of lips, 296
Early stage of idiopathic atrophy; case for diagnosis, 81
Mycosis fungoides or extracellular cholesterosis, 148

Occupational Diseases: See under Industry

Ochronosis, 66

Odors, allergy to food odors; its relation to management of infantile eczema, 62

Ohmart, L. M.: Coal tar in dermatologic preparations, *421
New protective cream, *436

Oils, dermatitis from cutting oils, *179

Ointments: See also Skin, diseases
coal tar in dermatologic preparations, *421
rational pharmaceutic treatment of diseases of skin, *16
studies on ointments containing chrysarobin, *1
studies on ointments; zinc oxide pastes, *8

Oliver, E. A.: Nevus striatus symmetricus unguis; report of case with involvement of thumbs and little fingers, *190

Omens, D. V.: Amyloidosis cutis or lichen simplex chronicus, 219

Omens, H. D.: Amyloidosis cutis or lichen simplex chronicus, 219
Scleroderma (extensive unilateral morphea of band type), 214

Onychia: See Nails

Onycholysis: See Nails

Oppenheim, M.: Generalized progressive scleroderma, 215
Local arterial embolism, 219
Pityriasis lichenoides chronica; case for diagnosis, 284

Optic Disk, Choked: See Neuritis, optic

Oriental Sore: See Leishmaniasis

Oroya Fever, Carrion's disease; immunologic studies, 199

Ossification and calcification of steatomas of scrotum; report of case, *12; correction, 304

Ostwald, E.: Dermatitis due to hair lacquer, *136

Otorhinolaryngology, vitamin therapy today, 282

Otsuka, M.: Acrodermatitis chronica atrophicans, 216
Paraffinoma, 216
Sarcoidosis, 213

Oxygen consumption of skin and hair growth after adrenalectomy in white rat, 199

Pachyonychia: See Nails, abnormalities

Paget's Disease: See Skin

Palsy: See Paralysis

Panniculitis and lupus erythematosus of cheeks, 142

Papilledema: See Neuritis, optic

Papillomas, effect of roentgen rays on cell-virus associations, 281

Paracoccidioides: See Blastomycosis

Paraffinoma, 216

Parakeratosis scutularis or psoriasis, 211

Paralysis, facial; Bell's palsy and herpes zoster, 445
General: See Dementia Paralytica
motor complications of herpes zoster, 139
Weber's syndrome of syphilitic origin, 64

Paraphenylenediamine, dermatitis due to, *177

Parapsoriasis, 293
en plaques, 204
en plaques or mycosis fungoides, 211
guttata; case for diagnosis, 203
(Haberman?); case for diagnosis, 79
mycosis fungoides simulating parapsoriasis, 74
pityriasis lichenoides chronica; case for diagnosis, 284
pityriasis lichenoides et varioliformis acuta (Habermann), 292
psoriasiform eruption (psoriasis, tertiary syphilis or parapsoriasis en plaques), 383
varioliformis; case for diagnosis, 73

Pardo-Castello, V.: Rational pharmaceutic treatment of diseases of skin, 223

Paresis: See Dementia Paralytica

Parrot's Disease: See Pseudoparalysis

Pascher, F.: Tuberculin patch test and Mantoux test; comparative study in cases of various dermatoses, including tuberculoderms, *256

Pastes, studies on ointments; zinc oxide pastes, *8

Patch Test: See Anaphylaxis and Allergy

Peck, S. M.: Dermatitis from carrots, *266

Pediculi, delousing of clothing by fumigation, 282
time required for eggs of body louse (Pediculus humanus corporis De Geer) to develop and hatch at different temperatures, 201

Pellagra: See also Riboflavin
asymmetric cutaneous lesions in, *335
treatment with nicotinic acid, 141

Pemphigus: See also Epidermolysis bullosa
atypical lupus erythematosus or Senear-Usher syndrome, 297
benign chronic, or dermatitis herpetiformis, 362
chronic benign; case for diagnosis, 458
conjunctivae with scarring of skin; report of 3 additional cases, *113
erythematosus; case for diagnosis, 82
erythematosus, pemphigus vulgaris or dermatitis herpetiformis, 221
erythematosus (Senear-Usher), 298, 455
ocular, 362
vulgaris, 295, 363
vulgaris; danger of cutaneous reaction to sulfonamides; report of 2 cases of bullous eruption following use of sulfanilamide, 1 of which resembled pemphigus vulgaris, 63
vulgaris, localized, or erythema multiforme bullosum, 364
vulgaris, pemphigus erythematosus or dermatitis herpetiformis, 221
vulgaris (recovered), 383

Penis, balanitis xerotica obliterans, 299, 304
balanitis xerotica obliterans; report of case, *118
balanosclerosis with beginning epithelioma, 205
chancriform penile lesions, 80, 148
gumma of; case for diagnosis, 210
leukoplakia with early epitheliomatous changes, 286
relationship of balanitis xerotica obliterans to lichen sclerosus et atrophicus, *57

Periarteritis nodosa, 372
nodosa or erythema nodosum, 298

Perifolliculitis abscedens et suffodiens, 154
capitis abscedens et suffodiens (due to use of "Teek," hair straightener), 67

Perlson, J.: Therapeutic malaria for patient who had undergone splenectomy, *137

Permanent Waving: See Cosmetics

Persky, B. P.: Multiple idiopathic hemorrhagic sarcoma of Kaposi in full-blooded Negro, *270

Perspiration: See under Sweat Glands

Petrolatum, cutaneous absorption; value of petrolatum, anhydrous wool fat and other bases in percutaneous absorption of topically applied cottonseed allergen, *427

Pfahler, G. E.: Cavernous hemangioma, 369

Phalanges: See Fingers and Toes

Photosensitization: See under Light

Phthirus Pubis: See Pediculi

Pigmentation: See also Hair; Nevi; Ochronosis; Xeroderma pigmentosum; etc.
greenish pigmentation of nail plates from Bacillus pyocyaneous infection; report of 2 cases, *136
hyperpigmentation (arsenic?), 70
pigmented lesion of cheek, 65
Pinkus, H.: Acid orcein-Giemsa stain (modification of Unna-Taenzer method); useful routine stain for dermatologic sections, *355
Pinta (mal del pinto, carate) in continental United States, 62
White: See Vitiligo
Pituitary Preparations, melanotropic hormone and vitiligo, 200
Pityriasis, Lichenoides: See Parapsoriasis
rosea; treatment by injection of typhoid vaccine, 359
rubra pilaris, 69, 301
Plants: See also under names of plants, as Manzanillo Tree; etc.
dermatitis caused by representatives of Anacardiaceae in tropical countries, 359
dermatitis from Semecarpus anacardium (bhilawanol of marking nut) spread by mail, 200
dhobie mark dermatitis, 200
Poikiloderma, 376
amyloidosis cutis or lupus erythematosus, 209
atrophicans vasculare, 85
of Civatte, 448
report on case (Civatte?), 146
Poisons and Poisoning: See Dyes; and under names of substances, as Mercury; etc.
Pollen: See Hay Fever
Polycythemia and erythromelalgia, 293
Popper, H.: Blood vitamin A and cutaneous diseases, *103
Poradenitis: See Lymphogranuloma Venereum
Porokeratosis: See Keratosis
Port Wine Mark: See Nevi flammeus
Potassium Chloride: See Eczema
Pregnancy, erythema palmare, 451
herpes gestationis; case for diagnosis, 209
Proteins, histamine-protein complexes; synthesis and immunologic investigation; histamine-azoprotein, 443
in Blood: See Blood, proteins
Prurigo, 371
Pseudoparalysis, Parrot's, 444
Pseudopelade: See Alopecia cicatrisata
Psoriasis and eye color, 360
of nails, 301
or parakeratosis scutularis, 211
or squamous eczema, 301
psoriasiform eruption (psoriasis, tertiary syphilis or parapsoriasis en plaques), 383
vulgaris; mycosis fungoides preceded by psoriasis vulgaris of 30 years' duration, 143
Pumice, clinical observations on use of soaps containing abrasive, *188
Purpura, acute lupus erythematosus or acute thrombocytopenia, 205
hemorrhagic, due to toxic effects of quinine salts, 445
Pyoderma gangrenosum; multiple disseminated sphaceloderma with generalized amyloidosis, 76
Kaposi's varicelliform eruption, 200
Ulcerosum Tropicalum: See Ulcers
varicelliform eruption of Kaposi, 444
Pyodermitis: See Dermatitis vegetans

Quinine, hemorrhagic purpura due to toxic effects of quinine salts, 445

Races: See Indians, American; Negroes; etc.
Radiations: See Radium; Roentgen Rays; etc.
Radiodermatitis: See under Radium; Roentgen Rays
Radium, chronic radiodermatitis resulting from treatment of port wine mark with radium, 153
Therapy: See Hemangioma
Ral Tree: See under Plants
Rambousek, E. S.: Sarcoidosis, 446
Rappaport, rapid macroflocculation test for syphilis; method of Rappaport and Eichhorn, *240
Rattner, H.: Fungous infection or mycosis fungoides, 218
Granuloma annulare or necrobiosis lipoidica diabeticorum, 284
Hidradenitis suppurativa, 285
Raynaud's Disease with extensive scleroderma, 387
von Recklinghausen's Disease: See Neurofibromatosis
Reif, F. B.: Balanitis xerotica obliterans, 304
Reiss, F.: Successful inoculations of animals with Trichophyton purpureum; observations on course of disease and immunologic and histologic features, *242
Reiter's Disease: See Arthritis; Conjunctivitis
Resins, contact dermatitis due to hair lacquer, 454
dermatitis due to hair lacquer, *136
dermatitis due to resin-finished shorts and fabrics; investigation of cause, pathogenesis and related phenomena observed in 10 cases, 359
hair lacquer pad dermatitis, *198
Respirator: See Masks
Retinitis, primary syphilis treated by 26 week course of mapharsen and bismuth; acute basilar meningitis with neuroretinitis developing during treatment, *109
Riboflavin: See also Pellagra; Vitamins, B
ocular criteria of deficiency of, 283
Ricchiuti, J. F.: Epidermodysplasia verruciformis, 365
Rickettsia: See Rocky Mountain Spotted Fever
Ringworm: See also Microsporosis; Mycosis; Tinea; Trichophytosis; etc.
fungous infections of foot, 442
of scalp; successful use of roentgen rays to epilate local areas of infection, *107
rational pharmaceutic treatment of diseases of skin, 223
Ritter's Disease: See Dermatitis, exfoliative
Robinson, S. S.: Colloid degeneration of skin (colloid milium), 454
Early stage of idiopathic atrophy; case for diagnosis, 81
Localized scleroderma, 455
Rocky Mountain Spotted Fever, 139
Roentgen Rays, calibration of x-ray equipment for superficial therapy, *250
effects on cell-virus associations, 281
epithelioma of cheek developing 15 years after tricho x-ray treatment for hair on face, 443
protection in radiology; exhibit, 201
ringworm of scalp; successful use of roentgen rays to epilate local areas of infection, *107
rodent ulcer of breast following radiodermatitis, 205
Therapy: See under names of organs, regions and diseases, as Cancer; Epithelioma; Melanoma; Papilloma; etc.
Roentgenotherapy, area factor in roentgen irradiation, *346
Rogers, J. D.: Melanoma, 457
Pseudopelade, 292
Rosacea-like tuberculid (of Lewandowsky), 296
Subacute disseminate lupus erythematosus, 454
Ronchese, F.: Calcification and ossification of steatomas of scrotum; report of case, *12; correction, 304
Rosenbach Disease: See Erysipeloid
Rosenthal, L. H.: Psoriasis or squamous eczema, 301
Rothman, S.: Acrodermatitis chronica atrophicans, 216
Epidermolysis bullosa, 285
Keratoma dissipatum palmoplantare (Brauer) (keratoderma papulosum), 217
Pemphigus vulgaris, pemphigus erythematosus or dermatitis herpetiformis, 221
Rule, A. M.: Sulfonamide compounds in treatment of Erysipelothrix rhusiopathiae infections: effectiveness of sulfanilamide, sulfapyridine, sulfathiazole and sulfadiazine against experimental infection in mouse and against erysipeloid of Rosenbach in man, *27
Rulison, R. H.: Hyperpigmentation (arsenic?), 70

Sabetta, A.: Hodgkin's disease of skin, 289

Sack, S. S.: Cutaneous absorption; value of petrolatum, anhydrous wool fat and other bases in percutaneous absorption of topically applied cottonseed allergen, *427

Sagher, F.: Fixed erythema due to sulfanilamide with gradually lessening sensitivity, *418

Salmonella cholerae suis as cause of indolent subcutaneous abscess, 359

Sarcoidosis, 208, 213, 382, 446
cutaneous tuberculosis (sarcoids, tuberculids or lupus vulgaris?), 220
lupus erythematosus or lichenoid sarcoid, 68
sarcoid, 302
sarcoid; case for diagnosis, 284
tuberculosis of skin; sarcoid (?), 364
tuberculosis of skin (sarcoidosis?), 206

Sarcoma: See also Cancer; Tumors; and under names of tumors, as Lymphosarcoma; etc.
multiple idiopathic hemorrhagic (Kaposi), 295
multiple idiopathic hemorrhagic, of Kaposi in full-blooded Negro, *270

Scalp, dermatitis herpetiformis limited to, 453
Diseases: See under names of diseases, as Ringworm; Tinea; etc.
folliculitis capitis abscedens et suffodiens associated with tinea capitis, 364
massive fibroma of, 138
perifolliculitis capitis abscedens et suffodiens (due to use of "Teek," hair straightener), 67
ringworm of scalp; successful use of roentgen rays to epilate local areas of infection, *107
turban tumor, 147

Scars: See Cicatrix

Schaumann's Disease: See Sarcoidosis

Schildkraut, J. M.: Balanosclerosis with beginning epithelioma, 205
Parapsoriasis en plaques, 204

Schools, vitamins and physiologic function, 283

Scleredema: See Scleroderma

Sclerema: See Scleroderma

Scleroderma, bullous lichen sclerosus et atrophicus; its relation to bullous scleroderma, *423
circumscribed, 451
circumscribed, associated with lichen sclerosus et atrophicans, 386
early, or acrodermatitis chronica atrophicans, 153
extensive unilateral morphea of band type, 214
extensive, with Raynaud's syndrome, 387
generalized progressive, 210, 215
healed, 451
localized, 455
morphea; case for diagnosis, 207
morphea (for therapeutic suggestions), 384
scleredema adultorum, 452

Scrofuloderma: See Tuberculosis

Scrotum, calcification and ossification of steatomas; report of case, *12; correction, 304

Scull, R. H.: Erythrasma, 285
Leukoplakia of penis with early epitheliomatous changes, 286

Sebaceous Glands, calcification and ossification of steatomas of scrotum; report of case, *12; correction, 304

Self Mutilation: lupus erythematosus complicated by dermatitis factitia, 456
lupus erythematosus or dermatitis factitia, 452

Semecarpus Anacardium: See under Plants

Senear, F. E.: Epidermolysis bullosa or lupus erythematosus, 287
Moeller's glossitis, 212

Senear-Usher Syndrome: See Pemphigus

Septicemia: See Bacteria, erysipelothrix

Sex, xeroderma pigmentosum; report of case and consideration of incomplete sex linkage in inheritance of disease, *157

Shaffer, B.: Granuloma annulare of lip, 289

Shaffer, L. W.: Concentration of arsenic in blood after administration of mapharsen by rapid drip method; clinical and experimental studies, *321

Sharlit, H.: Circumscribed scleroderma associated with lichen sclerosus et atrophicans, 386
Ulceration of toe on nevus flammeus; case for diagnosis, 152

Shaw, C.: Dermatitis due to shoes, *191

Shelton, J. M.: Replacement graft in surgical treatment of lupus vulgaris, *60

Shipyards: See Industry

Shoes, dermatitis due to, *191

Shorts: See Clothing

Sidlick, D. M.: Pemphigus vulgaris, 363

Silverman, M.: Gumma of penis; case for diagnosis, 210
Sarcoidosis, 208
Tertiary syphilis of skin or multiple epithelioma of skin, 206

Silvers, S. H.: Microsporon audouini infection of eyelashes, *436
Morphea; case for diagnosis, 207

Skeleton: See Bones

Skin: See also Dermatology
Abnormalities: See also Atrophy; Ectodermal Defect; Pigmentation; etc.
abnormalities; congenital defect in newborn infant, 280
abnormalities; cutis hyperelastica (Ehlers-Danlos syndrome), 150
acid orcein-Giemsa stain (modification of Unna-Taenzer method); useful routine stain for dermatologic sections, *355
amyloidosis of, 143
asymmetric cutaneous lesions in pellagra, *335
Atrophy: See Atrophy
Cancer: See Cancer; Epithelioma; Sarcoma; etc.
changes of nutritional origin, 282
clinical observations on use of soaps containing abrasive, *188
coal tar in dermatologic preparations, *421
cutaneous absorption; value of petrolatum, anhydrous wool fat and other bases in percutaneous absorption of topically applied cottonseed allergen, *427
cutaneous autosensitization; role of staphylococci in chronic eczema of hands, *124
cutaneous manifestations of circulatory disorders of foot, 442
dermal myiasis; report of 3 cases, *309
Diseases: See also Dermatitis; and under names of diseases, as Eczema; Herpes; Mycosis; etc.; and under names of plants and drugs, as Arsphenamines; etc.
diseases; Alibour's water, 357
diseases; aloe vera plant, *46
diseases and blood vitamin A, *103
diseases; area factor in roentgen irradiation, *346
diseases; colloid degeneration (colloid milium), 454
diseases; colloid degeneration; report of 3 cases from Australia, *234
diseases; Dalibour's water; its uses in dermatology, 63
diseases; dermatologic lesions about eyes, 138
diseases; dermatologic significance of tissue eosinophilia, *19
diseases; dermatology in Army Station Hospital, *91
diseases; external use of 30 to 50 per cent sulfur in petrolatum in various dermatoses, 63
diseases; functional factors in common dermatoses, 139
diseases; local use of sulfonamide compounds in dermatology, 140
diseases; necrobiosis lipoidica diabeticorum, 206
diseases; necrobiosis lipoidica diabeticorum (numerous lesions of lower extremities), 212
diseases; necrobiosis lipoidica diabeticorum or granuloma annulare, 284
diseases; necrosis and necrobiosis lipoidica diabeticorum, 370
diseases; nutritional dermatoses in rat; comparison of disseminated neurodermatitis and experimental magnesium deficiency, *33; correction, 226
diseases, rational pharmaceutic treatment, *16, 223

Skin—Continued
diseases; tuberculin patch test and Mantoux test; comparative study in cases of various dermatoses, including tuberculoderms, *256
diseases; use of Dalibour's water in treatment, 63
epidemic in children characterized by diversity of lesions in skin and mucous membranes, probably caused by Streptococcus pyogenes, 360
extramammary Paget's disease, 281
gold dermatitis with spectrographic evidence of deposition of gold in skin, 365
Hodgkin's disease of, 289
Hodgkin's disease with cutaneous manifestations, 457
hydroa estivale with secondary cutaneous changes, 84
increased resistance to viral infection as result of increased fluid in tissues, 200
Inflammation: See Dermatitis
inflammatory disorders of skin of feet, 442
oxygen consumption of skin and hair growth after adrenalectomy in white rat, 199
Pigmentation: See under Pigmentation
primary gonorrheal cutaneous infection, 61
relation of pyogenic skin infections to skin-carrier rate, 282
replacement graft in surgical treatment of lupus vulgaris, *60
studies in sensitization to skin; production of antibodies to skin by means of synergistic action of homologous skin antigen and staphylococcus toxin, 200
studies on ointments containing chrysarobin, *1
studies on ointments; zinc oxide pastes, *8
Syphilis: See Syphilids
Tuberculosis: See Tuberculids; Tuberculosis
ulcerative diphtheria of; sporadic and epidemic types observed in Haifa, Palestine, *408
Ulcers: See Ulcers

Smallpox, chronic lymphatic leukemia, complicated by vaccination, 288
increased resistance to viral infection as result of increased fluid in tissues, 200
Kaposi's varicelliform eruption, 200
lymphosarcoma, complicated by vaccination, 288
nosocomial transmission of eczema vaccinatum, 64
varicelliform eruption of Kaposi, 444

Snow, J. S.: Dermatitis venenata and keratoconjunctivitis caused by manzanillo tree, *236

Soaps, clinical observations on use of soaps containing abrasive, *188

Societies, American Board of Dermatology and Syphilology, *133, 304
dermatologic, directory of, 87
foreign, directory of, 87
local, directory of, 89
national, directory of, 87
sectional, directory of, 87
Society for Investigative Dermatology, 304
state, directory of, 88

Society Transactions:
Atlantic Dermatologic Conference, 362
Brooklyn Dermatological Society, 206
Chicago Dermatological Society, 212, 284
Cleveland Dermatological Society, 446
Los Angeles Dermatological Society, 79, 292, 453
Los Angeles Dermatological Society and San Francisco Dermatological Society, 146
Manhattan Dermatologic Society, 152, 385
Meeting of Civilian and Military Dermatologists Practicing or Serving in Hawaii, 297
Minnesota Dermatological Society, 449
New York Academy of Medicine, Section of Dermatology and Syphilis, 74, 150, 374; correction, 304
New York Dermatological Society, 65, 142
Philadelphia Dermatological Society, 202, 287, 381
San Francisco Dermatological Society, 302

Sodium Gold Thiosulfate: See Gold and Gold Compounds

Sompayrac, L.: Lupus erythematosus with false positive Kahn reactions, *355

Sore, Oriental: See Leishmaniasis

Sphaceloderma, multiple disseminated sphaceloderma with generalized amyloidosis, 76

Spies, T. D.: Asymmetric cutaneous lesions in pellagra, *335

Spirochaeta Pallida: See also Syphilis
cultivation of Treponema pallidum with preservation of virulence, 61
morphology of Treponema pallidum in electron microscope; demonstration of flagella, 138

Splenectomy, therapeutic malaria for patient who had undergone splenectomy, *137

Spolyar, L. W.: Dermatitis from carrots, *266

Sporotrichosis, 454
effectiveness in vitro of sulfonamide compounds on Sporotrichum schencki; report of 5 cases of sporotrichosis in North Carolina, *305

Stains and Staining, acid orcein-Giemsa stain (modification of Unna-Taenzer method); useful routine stain for dermatologic sections, *355

Staphylococci, cutaneous autosensitization; role of staphylococci in chronic eczema of hands, *124
effect of actinomycin, clavacin and tyrothricin on staphylococcal toxin, 62
studies in sensitization to skin; production of antibodies to skin by means of synergistic action of homologous skin antigen and staphylococcus toxin, 200

Steatoma, calcification and ossification of steatomas of scrotum; report of case, *12; correction, 304

Steigmann, F.: Blood vitamin A and cutaneous diseases, *103

Stevens-Johnson Disease: See Erythema multiforme

Stilbestrol: See Estrogens

Stoklosa, M. J.: New protective cream, *436

Stomatitis, aphthous; Behcet's syndrome with involvement of central nervous system; report of case, with necropsy, of lesions of mouth, genitalia and eyes; review of literature, *227
Vincent's: See Fusospirochetosis

Strakosch, E. A.: Studies on ointments; ointments containing chrysarobin, *1
Studies on ointments; zinc oxide pastes, *8

Streptococci, epidemic in children characterized by diversity of lesions in skin and mucous membranes, probably caused by Streptococcus pyogenes, 360

Sudoriparous Glands: See under Sweat Glands

Sulfanilamide: See under Sulfonamides

Sulfapyridine: See Sulfonamides

Sulfonamides, cutaneous hypersensitivity to, 64
danger of cutaneous reaction to; report of 2 cases of bullous eruption following use of sulfanilamide, 1 of which resembled pemphigus vulgaris, 63
effectiveness in vitro of sulfonamide compounds on Sporotrichum schencki; report of 5 cases of sporotrichosis in North Carolina, *305
erythema nodosum due to sulfathiazole? 380
erythema nodosum following sulfathiazole therapy, 155
fixed erythema due to sulfanilamide with gradually lessening sensitivity, *418
local use of sulfonamide compounds in dermatology, 140
lupus erythematosus (initiated by sulfanilamide?), 202
misuse of sulfonamide compounds, *249
sensitivity to topical application of sulfathiazole ointment, 442
sulfathiazole eruptions; sensitivity induced by local therapy and elicited by oral medication, 200
Therapy: See Acrodermatitis continua; Actinomycosis; Blastomycosis; Chancroid; Erysipeloid; etc.

Sulfur: See Skin, diseases

Sullivan, M.: Nutritional dermatoses in rat; comparison of disseminated neurodermatitis and experimental magnesium deficiency, *33; correction, 226

Sultzaberger, J. A.: Concentration of arsenic in blood after administration of mapharsen by rapid drip method; clinical and experimental studies, *321

Sulzberger, M. B.: Tuberculin patch test and Mantoux test; comparative study in cases of various dermatoses, including tuberculoderms, *256

Sunburn: See Light

Sunlight: See Light

Suprarenals: See Adrenals

Sutton's Disease: See Vitiligo

Sweat Glands, hereditary ectodermal dysplasia of anhidrotic type, presenting conic tapering of hands, various osseous anomalies and atopic eczema, 371
hereditary ectodermal dysplasia; report of case, with experimental study, *410
hidradenitis suppurativa, 285

Sweitzer, S. E.: Epithelioma of anus, 450
Erythema palmare, 451
Healed scleroderma, 451
Lichen chronicus simplex associated with hypoestrogenic keratoderma, 451
Lichen planus ocreaformis, 450
Livedo reticularis, 450
Subungual exostosis, 451

Syphilids, frambesiform (iododerma, bromoderma and blastomycosis not ruled out), 290
papulonecrotic tuberculid in Negro; its clinical resemblance to acne vulgaris and early syphilis, *351

Syphilis: See also Chancre; Neurosyphilis; Spirochaeta Pallida; Syphilids; Venereal Diseases; and under names of organs and regions, as Liver; etc.
arsenoresistant (improved), 150
arsenoresistant, pyogenic ulceration and destruction of nose, 77
concentration of arsenic in blood after administration of mapharsen by rapid drip method; clinical and experimental studies, *321
congenital, antiquity of, 441
congenital, cardiovascular involvement in, 61
congenital, in woman with exaggerated saber skins and (recently) active gummatous ulceration, 366
early; results of 20-week treatment schedule, 61
Hereditary: See Syphilis, congenital
intensive ambulatory therapy; 30 day mapharsen technic, *403
intensive methods of treating, 139
lupus erythematosus with false positive Kahn reactions, *355
Mazzini slide flocculation test; sensitivity to its antigen, 280
or lupus erythematosus, 218
oral administration of mapharsen in treatment of experimental syphilis of rabbits, 62
postarsphenamine lichen planus, 61
primary and secondary syphilis; chronic lymphatic leukemia, 145
primary, treated by 26 week course of mapharsen and bismuth; acute basilar meningitis with neuroretinitis developing during treatment, *109
problem of treatment-resistant syphilis, 61
quantitative complement fixation test for syphilis in malaria-treated syphilis; effect of diluent, 358
rapid macroflocculation test; method of Rappaport and Eichhorn, *240
recurring erythematous papulonodular syphilis, 374
relation of blood ascorbic acid clearance to arsenical intolerance in syphilotherapy, 444
serologic change occurring during short courses of neoarsphenamine and bismuth, such as were possible while patients stayed at venereal diseases center for African soldiers, 361
symmetric syphilitic granuloma of elbows or Bowen's disease of ankle, 289
syphilitic aneurysm of celiac artery, 280
tertiary; benign neoplasm of tongue (gumma?), 303
tertiary; diffuse gummatous myocarditis, 199
tertiary; gumma of penis; case for diagnosis, 210
tertiary, of skin or multiple epithelioma of skin, 206
tertiary; psoriasiform eruption (psoriasis, tertiary syphilis or parapsoriasis en plaques), 383

Syphilis—Continued
toxicity and therapeutic effectiveness of mapharsen by intramuscular administration, 62
treatment of early and latent syphilis, 280
unusually high icterus index in patient with fatal hepatic necrosis following mapharsen, 359
verification test in serology of, 281
Weber syndrome of syphilitic origin, 64

Syphilology; American Board of Dermatology and Syphilology, *133, 304

Syringomyelia, familial, 139

Tabes Dorsalis: See also Neurosyphilis
postural hypotension in, 358

Tattooing, generalized mercurial (cinnabar) reaction following tattooing, *172

Tavs, L.: Mycosis fungoides (relationship to congenital ichthyosiform erythroderma), 366

Teek: See under Hair

Teeth, urticaria following dental silver filling, 443

Temperature: See also Cold; Heat
time required for eggs of body louse (Pediculus humanus corporis De Geer) to develop and hatch at different temperatures, 201

Templeton, H. J.: Gonorrhea of skin, *436

Thiobismol: See Bismuth and Bismuth Compounds

Thomas, C. C.: Gold dermatitis with spectrographic evidence of deposition of gold in skin, 365
Periarteritis nodosa, 372
Psoriasiform eruption (psoriasis, tertiary syphilis or parapsoriasis en plaques), 383
Tuberculous (?) ulcers, 369

Thrombocytopenia: See Purpura

Thyroid, association of lupus erythematosus and thyrotoxicosis in brother and sister, *111
hyperthyroidism, in association with localized myxedema, *415

Thyrotoxicosis: See under Thyroid

Ticks: See Rocky Mountain Spotted Fever

Tinea: See also Ringworm
capitis associated with folliculitis capitis abscedens et suffodiens, 364
capitis, epidemiology; study of tinea capitis in dispensary, *120
capitis (Trichophyton violaceum), 67
corporis with fluorescent lanugo hairs, 385

Tissue: See also Cells
dermatologic significance of tissue eosinophilia, *19
Staining: See Stains and Staining

Toe Nails: See Nails

Toes: See Fingers and Toes

Tolmach, J. E.: Folliculitis decalvans with lichen spinulosus, 151

Tongue, benign neoplasm (gumma?), 303
leukoplakia; results of treatment by electrodesiccation, 142
Moeller's glossitis, 212

Toxicodermas: See Dermatitis, venenata; Drugs, eruptions

Toxin: See under Staphylococci

Traub, E. F.: Erythema ab igni (from application of heat), 69
Keloids as sequel to varicella, *278
Lupus erythematosus, extensive discoid type, 72
Pigmented lesion of cheek, 65

Trauma, lupus erythematosus following, 365, 366
paratraumatic (pyogenic) eczema, 64

Treponema: See Spirochaeta Pallida

Trichophyton, generalized dermatophytosis (probably due to Trichophyton purpureum), 146
purpureum; fungous infection; case for diagnosis, 386
purpureum, successful inoculations of animals with; observations on course of disease and immunologic and histologic features, *242
violaceum; tinea capitis, 67

Trichophytosis: See also Mycosis
successful inoculations of animals with Trichophyton purpureum; observations on course of disease and immunologic and histologic features, *242

Trinitrotoluene: See Nitrotoluene
Tropical Medicine: See under names of tropical diseases, as Leishmaniasis; Ulcers; etc.
Tropics, dermatitis caused by representatives of Anacardiaceae in tropical countries, 359
Tryparsamide, effect of prolonged tryparsamide therapy on liver function, 61
Therapy: See Neurosyphilis
Tubercle Bacilli: See Tuberculosis
Tuberculids: See also under Tuberculosis
cutaneous tuberculosis (sarcoids, tuberculids or lupus vulgaris?), 220
papulonecrotic, in Negro; its clinical resemblance to acne vulgaris and early syphilis, *351
rosacea-like, of Lewandowsky, 296, 453
Tuberculin: See under Tuberculosis
Patch Test: See Tuberculosis
Tuberculoderms: See under Tuberculosis
Tuberculosis: See also Tuberculids; and under names of diseases, organs and regions, as Ulcers; etc.
cutaneous (sarcoids, tuberculids or lupus vulgaris?), 220
cutis; case for diagnosis, 384
hypothermia in experimental infections; effect of hypothermia on tuberculin sensitivity in guinea pigs, 62
lessened sensitivity to tuberculin in acne, *174
of skin; sarcoid (?), 364
of skin (sarcoidosis?), 206
quiescent tuberculoid leprosy, 300
tuberculin patch test and Mantoux test; comparative study in cases of various dermatoses, including tuberculoderms, *256
tuberculoid leprosy; case for diagnosis, 299
"visible tuberculin patch test," 280
Tumors: See also Angiokeratoma; Angioma; Cancer; Epithelioma; Fibroma; Hemangioma; Leiomyoma; Lymphoblastoma; Lymphosarcoma; Melanoma; Neurofibroma; Neuroma; Papilloma; Sarcoma; Steatoma; etc.; and under names of organs and regions, as Tongue; etc.
cutis hyperelastica (Ehlers-Danlos syndrome), 150
mycosis fungoides (tumor stage), 74
Typhoid Vaccine: See Pityriasis rosea
Tyrothricin: See Gramicidin

Ulcers: See also under names of organs and regions, as Breast; Genitals; etc.
arsenoresistant syphilis, pyogenic ulceration and destruction of nose, 77
congenital syphilis in woman with exaggerated saber skins and (recently) active gummatous ulceration, 366
multiple ulcerations of legs, 455
neural leprosy with mal perforans, 297
treatment of slow granulating sores and hypertrophic scars with zinc iontophoresis, 140
tropical, *260
tuberculous, 369
ulcerative diphtheria of skin; sporadic and epidemic types observed in Haifa, Palestine, *408
Ultraviolet Rays: See Light
Therapy: See under names of organs, regions and diseases
Umbilicus, gonorrhea of skin, *436
Underwear: See Clothing
Unna-Taenzer Method: See Stains and Staining
Urticaria following dental silver filling, 443
pigmentosa, 81
Usher-Senear Syndrome: See Pemphigus

Vaccination: See Smallpox
Varicella: See Chickenpox
Variola: See Smallpox
Venereal Diseases: See also Chancre; Chancroid; Gonorrhea; Neurosyphilis; Syphilis; etc.
problems in epidemiology in wartime, 358
Verruca, epidermodysplasia verruciformis, 83, 217, 365
plana (resistant to treatment), 65
treatment of plantar warts by formalin, 445
Vesication, bullous lichen sclerosus et atrophicus; its relation to bullous scleroderma, *423
lichen planus with vesicular lesions on buccal mucosa, 148
poikiloderma, 376
Vilter, R. W.: Asymmetric cutaneous lesions in pellagra, *335
Viruses: See also under Herpes; Lymphogranuloma Venereum; etc.
effect of roentgen rays on cell-virus associations, 281
Vitamins: See also Ascorbic Acid; Nicotinic Acid; Riboflavin
A: See also Carotene; Xerophthalmia
A; blood vitamin A and cutaneous diseases, *103
and physiologic function, 283
B: See also Nicotinic Acid; Riboflavin; Thiamine
B; vitamins for gray hair, 63
B_2: See Riboflavin
C: See Ascorbic Acid
skin changes of nutritional origin, 282
vitamin therapy today, 282
Vitiligo and melanotropic hormone, 200
clinical effects of paraaminobenzoic acid in, *132
leukoderma acquisitum centrifugum, 459
treated with gold tribromide orally, 293
Vollmer Patch Test: See Tuberculosis
Vulva, Esthiomene: See Lymphogranuloma Venereum
kraurosis vulvae, 79

Wallace, G. I.: Chlorophyll ointment in treatment of dermatophytosis of feet and similar lesions, *128
Walzer, A.: Acanthosis nigricans, 378
Cutaneous absorption; value of petrolatum, anhydrous wool fat and other bases in percutaneous absorption of topically applied cottonseed allergen, *427
Lichen nitidus, generalized, 381
Poikiloderma, 376
Psoriasis or parakeratosis scutularis, 211
Recurring erythematous papulonodular syphilis, 374
War: See also Military Medicine; etc.
dermatologists of United States and Canada in active service in present war, 437
need for more dermatologists in, 223
newer concepts in treatment of burns with suggestions for management of wartime thermal injuries, 444
problems in epidemiology of venereal disease in wartime, 358
Warts: See Verruca
Wassermann Reaction: See Syphilis
Water, Dalibour's: See Skin, diseases
Weber, L. F.: Epidermodysplasia verruciformis, 217
Epidermolysis bullosa or lupus erythematosus, 287
Weber's Syndrome: See under Paralysis
Weidman, F. D.: Cutaneous and systemic blastomycosis, 203
Welsh, A. L.: Clinical observations on use of soaps containing abrasive, *188
Wheal: See Urticaria
White Spot Disease: See Scleroderma
Winer, L. H.: Erythema palmare, 451
Wise, F.: Dermatitis artefacta, 71
Early scleroderma or acrodermatitis chronica atrophicans, 153
Knuckle pads, 144
Lupus erythematosus and panniculitis of cheeks, 142
Lupus erythematosus disseminatus, 72
Lupus erythematosus or lichenoid sarcoid, 68
Lupus vulgaris, 155
Lupus vulgaris of knee, 154
Mycosis fungoides preceded by psoriasis vulgaris of 30 years' duration, 143
Parapsoriasis varioliformis; case for diagnosis, 73
Raynaud's syndrome with extensive scleroderma, 387
Wolf, J.: Melanoma, 153

Wool Fat, cutaneous absorption; value of petrolatum, anhydrous wool fat and other bases in percutaneous absorption of topically applied cottonseed allergen, *427

Woolhandler, H. W.: Dermatology in Army station hospital, *91

Wright, C. S.: Acute lupus erythematosus or acute thrombocytopenia, 205
Erythema multiforme bullosum or localized pemphigus vulgaris, 364
Folliculitis capitis abscedens et suffodiens associated with tinea capitis, 364
Pachyonychia congenita, 368
Pityriasis lichenoides et varioliformis acuta (Habermann), 292
Tuberculosis of skin; sarcoid (?), 364
Tuberculosis of skin (sarcoidosis?), 206

Xanthoma diabeticorum; case for diagnosis, 292
diabeticorum (xanthoma eruptivum), 71
systemic, 149
tuberosum multiplex, 144

Xeroderma pigmentosum; report of case and consideration of incomplete sex linkage in inheritance of disease, *157

X-Rays: See Roentgen Rays

Yaws: See Frambesia

Young, J. W.: Dermal myiasis; report of 3 cases, *309

Zeisler, E. P.: Association of lupus erythematosus and thyrotoxicosis in brother and sister, *111
Melanoma, 220

Zinc, studies on ointments; zinc oxide pastes, *8
treatment of slow granulating sores and hypertrophic scars with zinc iontophoresis, 140

Zoster: See Herpes zoster

Zugerman, I.: Bullous lichen planus (?) cured by estradiol benzoate, 290
Necrobiosis lipoidica diabeticorum, 206
Necrosis and necrobiosis lipoidica diabeticorum, 370